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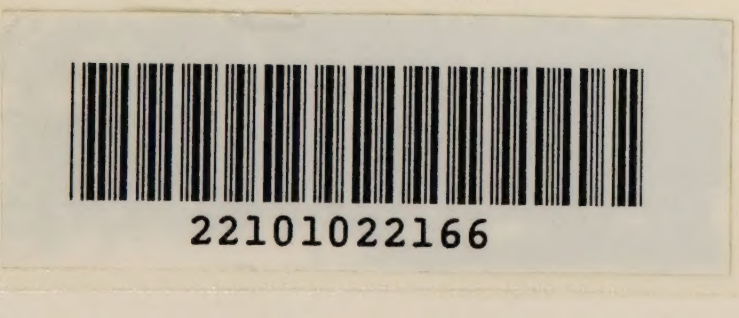

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CHAPTER VIII.

ON THE PATHOLOGY AND DIAGNOSIS OF DISEASES OF THE HEART AND LUNGS—OF THE THORAX GENERALLY—ITS CONTENTS AND PARIETES.

THE diseases of the heart and lungs are especially recognized and appreciated by *symptoms*, local and constitutional, as well as by so-called *physical signs;* and it is necessary to notice, in some preliminary sections, certain topics, a knowledge of which is essential to the accurate diagnosis of these diseases.

SECTION I.—RELATION OF THE THORACIC VISCERA TO THE WALLS OF THE CHEST.

(A.) *Regions of the Thorax.*

In order to enable the student and physician accurately to describe and record their observations, and more conveniently to communicate to others precise information respecting the seat and signs of internal diseases, it has been customary to map out the exterior surface of the chest and abdomen by lines into different compartments termed *regions.* These lines have not always been drawn in the same manner; and therefore, on the contrary, there has ever existed great discrepancy among writers and teachers in this and other countries respecting the number, the extent, and the names of the regions defined by such lines. The regions are quite arbitrary, and the lines described upon the surface are understood to correspond with imaginary planes passing towards the centre of the body. The points or *landmarks* on the surface through which these lines are drawn ought to be at once fixed points and obvious to the senses; so that the boundaries and contents of every region may be accurately defined, with the view of localizing physical signs as precisely as possible.

As it is necessary that a complete inspection of the body shall take in simultaneously the abdomen and the chest, the topography of these regions is generally given together; and

the annexed regional plans of the trunk are those devised by Mr. Paxton, of Oxford; and given by the late Sir John Forbes, in his excellent paper "On the Exploration of the Chest and Abdomen," in the first volume of the *Cyclopædia of Practical Medicine.** Prominent points of the skeleton are here made the basis of the system by which the regions are mapped out by vertical and horizontal lines. The descriptions of the boundaries and contents of the regions have been compiled on the authorities of Sibson, Walshe, Parkes, Lyons, Fuller, Quain, and Sharpey.

The vertical lines having relation to the chest are *eight* in number, and run as follows:—(1.) Along the middle of the sternum, from its upper to its lower end; (2.) From the acromial end of the clavicle to the external tubercle of the pubes (right and left); (4.) Along the spinous processes of the cervical and dorsal vertebræ; (5.) Along the posterior or spinal border of the scapulæ, from the clavicular transverse line to the mammary transverse line. (See figs. 5, 6, and 7.)

The horizontal or transverse lines are four in number, and are as follows:—(1.) Around the lower part of the neck, sloping downwards to the upper end of the sternum anteriorly, and to the last cervical vertebra posteriorly; (2.) Around the upper part of the chest in the line of the clavicles; (3.) Around the middle of the chest by the lower edge of the *third* rib, above the line of the male nipple, and touching the inferior border of the scapulæ behind; (4.) Around the lower part of

* To facilitate accuracy in this method of recording physical signs, blank outlines of the trunk of the body were first used by Piorry, and more recently they have been largely used both in clinical teaching and in recording cases for publication, by Professors Bennett, in Edinburgh, Gairdner, of Glasgow, and Beale, of London; the latter of whom has especially described the great advantages from their use, in the sixth number of the valuable *Archives* which he publishes (vol. ii., p. 97). The *outlines* merely of the trunk (without the dotted lines and the numbers), given in the text to illustrate the topography of the chest and abdomen, have been engraved on wood; so that the student can readily indicate the areas of physical signs by pencil, ink, or coloured chalk lines.

My publishers inform me that they are prepared to supply *fifty* blank copies of these outlines, at the cost of one shilling and sixpence, under the following title:—

"Outline Figures for Indicating the Areas of Physical Signs in the Clinical Diagnosis of Disease;" for the use of Students and Practitioners of Medicine. Arranged by WILLIAM AITKEN, M.D., &c. Charles Griffin & Co.

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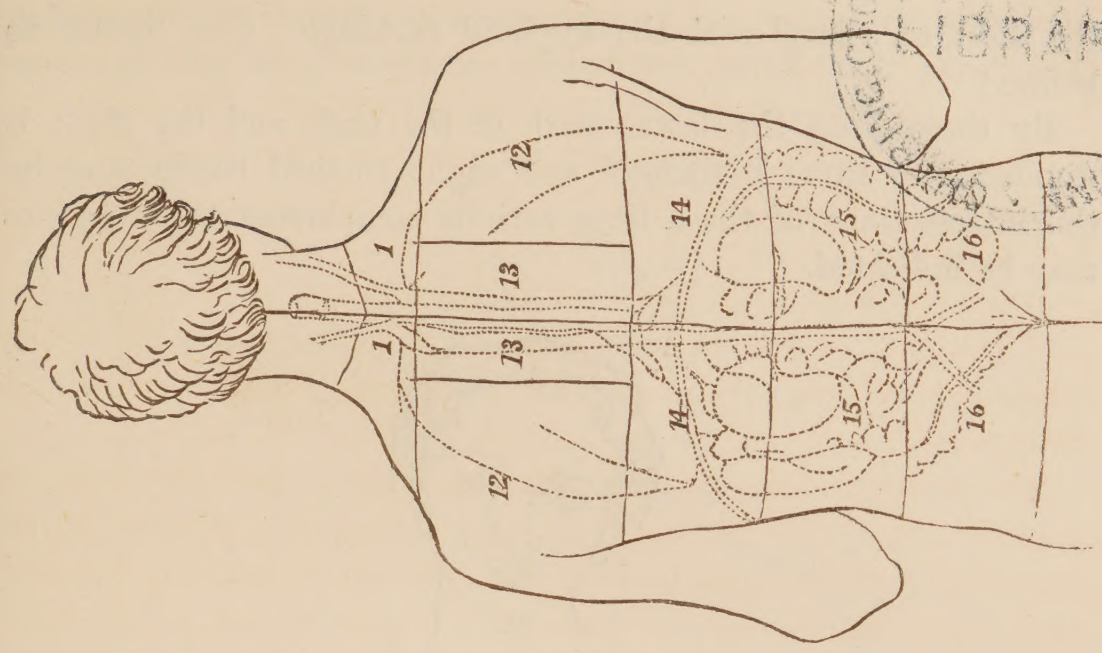

Fig. 7.

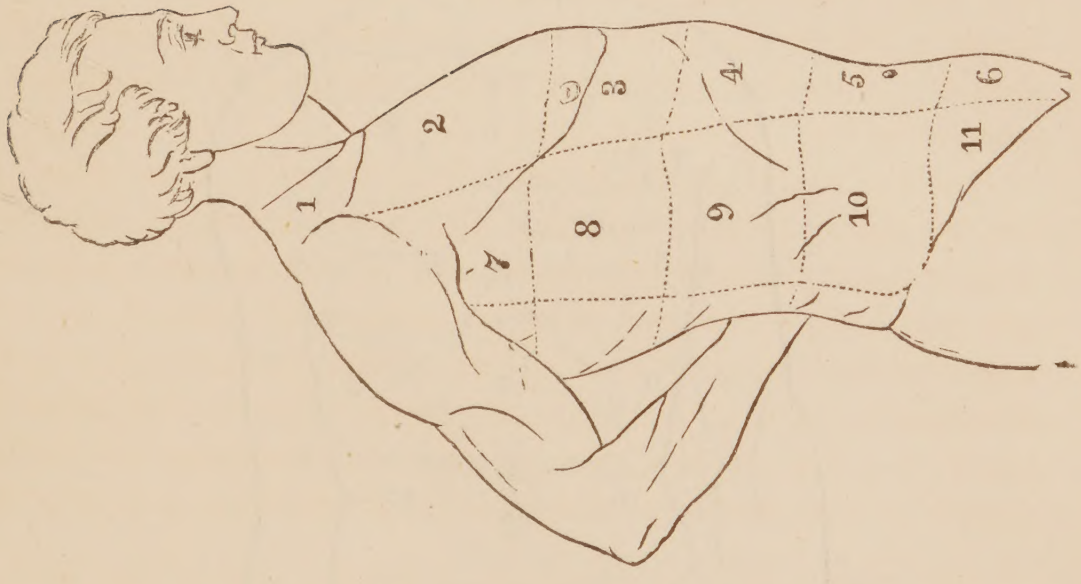

Fig. 6.

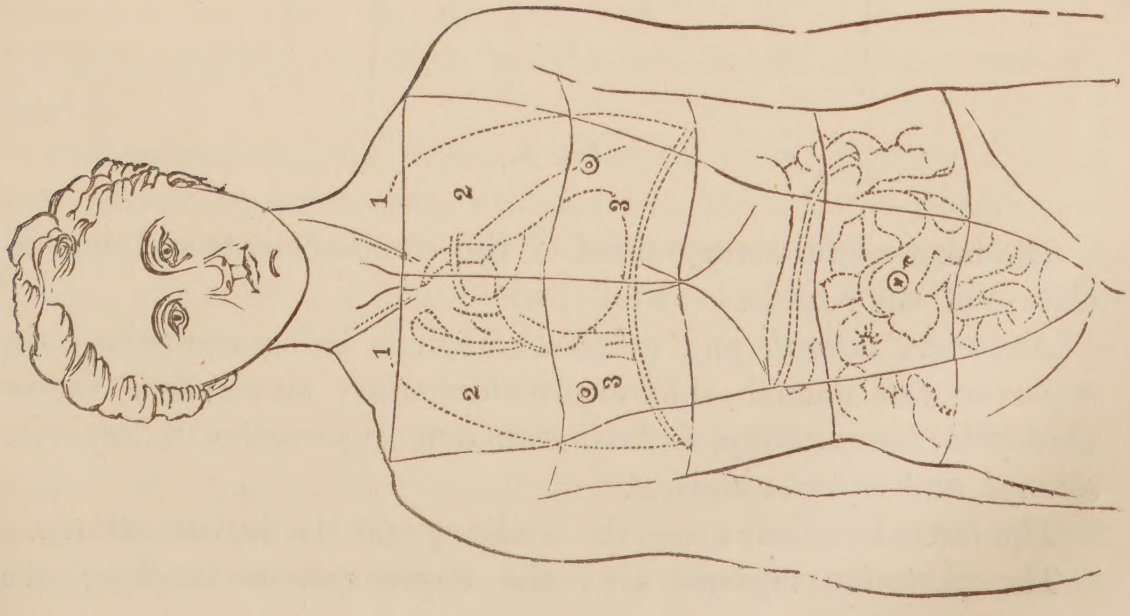

Fig. 5.

the chest, on a level with the xyphoid cartilage. (See figures as before.)

By these lines the lower part of the neck and the chest is divided into three horizontal and eight vertical bands; and by the intersections of these lines various compartments or regions may be indicated.

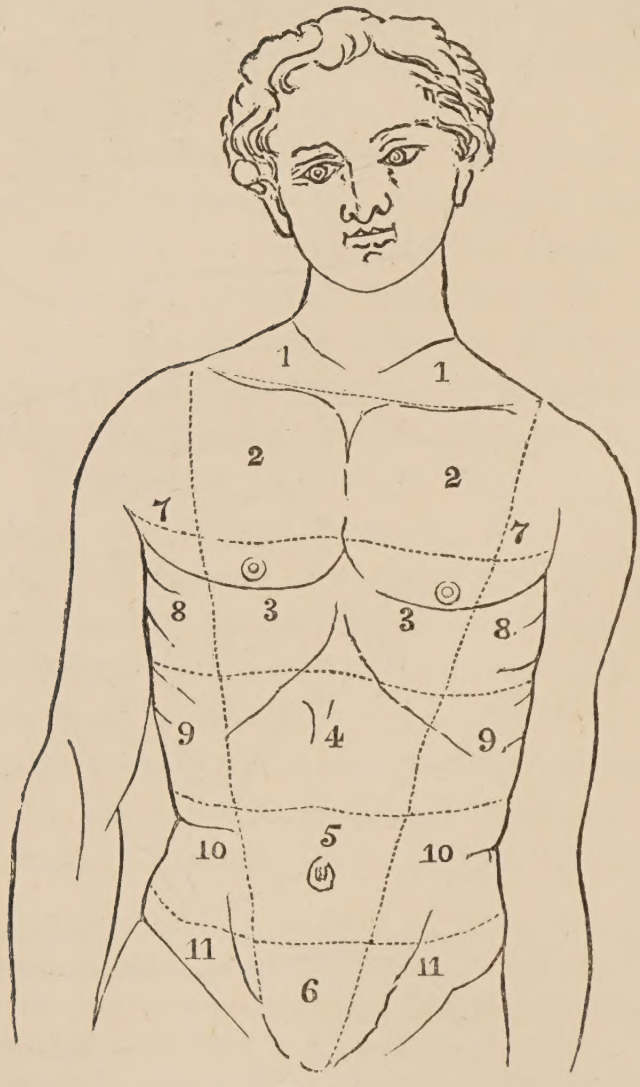

Fig. 8.

The most useful arrangement of the compartments and nomenclature of the regions is as follows:—

Anterior, lateral, and *posterior* regions being recognized, the *anterior* are named the *supra-clavicular, clavicular, infra-clavicular, mammary, infra-mammary, supra-sternal, superior sternal,* and *inferior sternal.*

The *lateral regions* are,—the *axillary* and the *infra-axillary.*

The *posterior regions* are,—the *supra-spinous region;* the

infra-spinous region (sometimes called the scapular); the *inter-scapular;* the *infra-scapular* (sometimes called the upper dorsal).

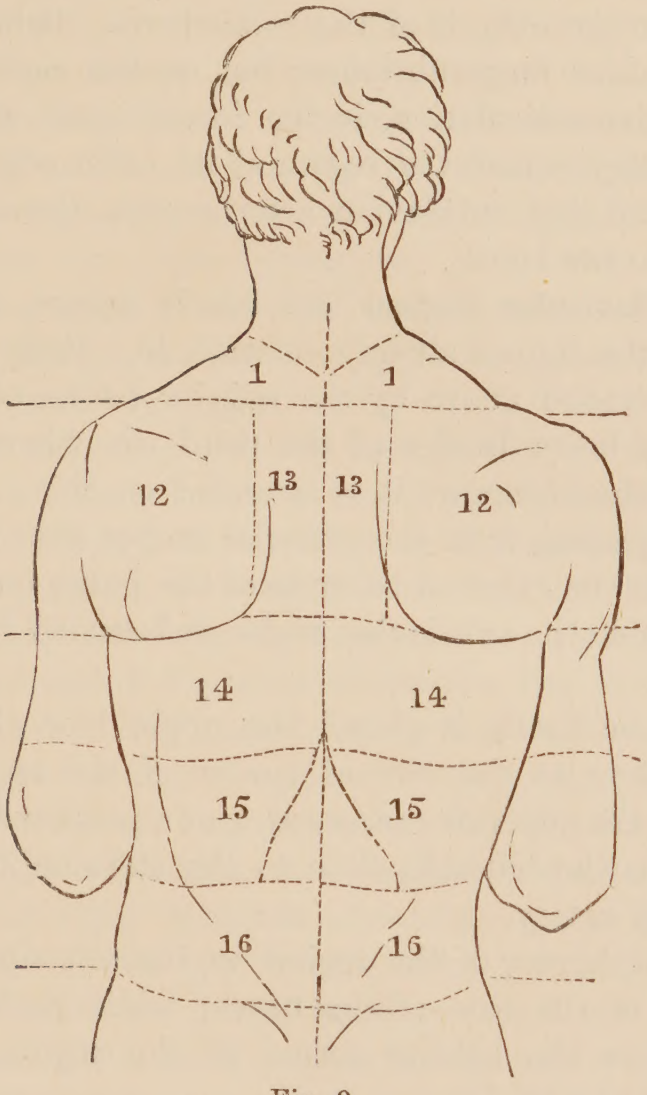

Fig. 9.

Of these regions the three sternal are single, all the rest are double.

The Supra-Clavicular Region is a small triangular space *above* the clavicle on either side, with its base internally at the trachea, its apex towards the outer end of the clavicle, and bounded below by the upper edge of that bone. A line drawn from the outer part of the clavicle to the upper rings of the trachea will limit its upper border.

In this region is found the triangular apex of the lung (figs. 8, 9), sometimes reaching on the right side a little higher than on the left, with portions of the subclavian and carotid arteries, and of the subclavian and jugular veins. The floor

of this region internally is formed by the upper surface of the first rib.

The Clavicular Region is very narrow and oblong, corresponding to the inner two-thirds of the collar-bone. Behind the bone lies on both sides lung-substance; but on the right side, at the sterno-clavicular articulation, is the *innominate artery*, and the subclavian artery crosses the region at its outer edge; on the left side the carotid and subclavian arteries pass upwards, almost at right angles to the bone.

The Infra-Clavicular Regions are nearly square, corresponding to No. 2 in the figures already referred to. Each region (right and left) is bounded above by the inferior border of the clavicle; below by the lower border of the third rib, where it joins the cartilages of the sternum; it is bounded on the outside by the vertical line passing from the acromial end of the clavicle downwards towards the external tubercle of the pubes (on either side); on the inner side the subclavian region is bounded by the edge of the sternum.

Within these limits is placed the upper lobe of the lung on both sides, close to the sternal border of the region. On the right side lie the superior *vena cava*, and a portion of the arch of the aorta. On the left side, close to the sternum, is the edge of the pulmonary artery.

The inferior border of the region on the left side corresponds to a portion of the base of the heart; while part of the right auricle occupies the inferior corner of the region towards the sternum on the right side.

The Mammary Region (No. 3, figs. 5, 6, and 7) has also a square-like form, and is bounded above by the line passing through the lower border of the *third* rib, where it joins the cartilages of the sternum below, by the line passing transversely on a level with the xyphoid cartilage (its upper border); outside by the vertical line passing to the outer tubercle of the pubes (on either side); and on the inner aspect by the edge of the sternum.

The contents of the mammary regions differ greatly on the two sides.

On the right side the lung lies throughout immediately underneath the ribs. Its inferior border turns off almost at right angles from the sternum, at the cartilage of the sixth rib,

whence its thin sharp border gently slopes outwards and downwards, so as to occupy the lower part of the region when the diaphragm is depressed. But when the diaphragm is elevated, the liver rises into this region up to the fourth interspace. The fissure between the upper and middle lobes of the right lung passes upwards and backwards obliquely across the region from about the *fourth* cartilage. The fissure between the middle and lower portion passes in the same direction from the *fifth* interspace. A portion of the right auricle, also a portion of the right and superior angle of the right ventricle, lie between the *third* and *fifth* ribs, close to the sternum, in the right mammary region.

On the left side, at about the level of the *fourth* cartilage, the anterior edge of the left lung passes obliquely downwards, having abruptly left the edge of its fellow on the opposite side, so as to expose a free space of variable size for the heart (fig. 10). The edge of the left lung thus reaches the *fifth* rib; whence it comes inwards and downwards to opposite the *sixth* rib or interspace, whence it finally passes nearly horizontally outwards and downwards into the lateral region. The anterior point of division of the lobes of the left lung lies about the fifth interspace, below the nipple. The left auricle and the left ventricle, with a small portion of the right ventricle about the apex, lie in the left mammary region; the apex of the heart generally lying immediately above the *sixth* costal cartilage (fig. 8 and fig. 11, under the description of the heart, a little farther on).

The Infra-Mammary Region has a somewhat triangular form. Its boundary alone is defined by the lower bounding line of the mammary region on each side: below, by the margins of the false ribs; inside, by the xyphoid cartilage or middle line; and outside, by the extension of the line from the acromial end of the clavicle to the outer tubercle of the pubes on either side.

On the right side this region contains the liver, with the edge of the lung overlapping it in front, to a variable extent during full inspiration.

On the left side the stomach and anterior edge of the spleen rise as high as the *sixth* rib in this region; and towards the inner portion of the region the left lobe of the liver lies in front of the stomach.

The Supra or Post-Sternal Region is a small hollow, bounded

below by the notch of the sternum, and laterally by the *sterno-mastoid* muscles.

The trachea almost completely fills this region; but on the right side the innominate artery lies at the lower angle; and in some persons the arch of the aorta reaches its lower border just at the notch of the sternum, where it may be felt pulsating. The region contains no lung.

The Upper or Superior Sternal Region comprehends that portion of the breast bone which is superior to the lower border of the *third* rib.

It covers the left and a small portion of the right innominate vein. The superior cava runs along its right edge; the ascending and transverse portions of the arch of the aorta; the pulmonary artery from its origin to its bifurcation; the aortic valves near the lower border of the *third* left cartilage at its junction with the sternum, or midway between the mesial line and the left edge of the sternum (the pulmonary valves being a little higher than these, more near the surface, and quite at the left of the sternum); and lastly, the trachea, with its bifurcation, on the level of the *second* ribs. The remains of the thymus gland, with areolar tissue and fat, lie in front of these parts, between the lateral pleural boundaries of the upper V-shaped portion of the anterior mediastinum, thus separating the edges of the lungs above towards their apices.

The Lower or Inferior Sternal Region comprehends the remainder of the sternum which lies below the level of the lower margin of the *third* rib. It contains the greater portion of the right ventricle, with the infundibulum of the pulmonary artery, and a small part of the left ventricle. The *mitral valve* is situated towards the upper end of this region, close to the left edge of the sternum, on a level with the *fourth* sterno-costal articulation; the *tricuspid valve* lies nearer the middle line, and more superficially. The edge of the right lung descends vertically along the middle line, and at the upper part of the region is a small portion of the left lung. Inferiorly and deeper seated is a portion of the liver, and sometimes of the stomach, while the line of union of the heart and liver corresponds with where the diaphragm intervenes.

The Axillary Region (figs. 5 and 6, No. 7) extends from the apex of the armpit above to the line continued which marks the inferior border of the infra-clavicular region. In front it is bounded by the posterior border of the infra-clavicular region, and

it extends to the external edge of the scapulæ behind on either side. The region can only be brought into view by lifting the arm over the head, or by carrying it away from the side (as in fig. 6). The region is hidden (as in fig. 5) when the arm is at rest by the side. It comprehends portions of the upper lobes of the lungs, a great volume of lung-substance, and, more deeply seated, large bronchi.

The Infra-Axillary Region (figs. 5 and 6, No. 8) is bounded above by the region already defined; anteriorly, by the mammary region; posteriorly, by the scapula; and below it extends to the margins of the ribs.

It contains on both sides the lower edge of the lung, sloping downwards from before to behind. On the right side, also, is the liver, between which and the walls of the chest is interposed a thin layer of lung-substance during a full inspiration. On the left side is the spleen and stomach.

The Supra-Spinous Regions have the same boundaries as the superior fossæ of the scapulæ, and correspond to the posterior surfaces of the apices of the lungs (figs. 7 and 9, No. 1).

The Infra-Spinous Region, sometimes called the Scapular (figs. 53, 7 and 9, No. 12). It is identical with the lower fossa of the scapula. It covers lung-substance.

The Inter-Scapular Region (right and left) lies between the inner margin of the scapulæ, divided into a right and a left region by the vertebral column, from the second to the sixth dorsal vertebra.

It covers lung-substance on each side of the mesian line, the main bronchi, and bronchial glands. On the left side is the œsophagus; and from the *third* or *fourth* vertebra downwards is the descending aorta. The bifurcation of the trachea takes place at the middle line between the two regions, but inclining rather to the right side (figs. 7 and 9, No. 13).

The Infra-Scapular or Lower Dorsal is bounded by the continuity of the transverse line, which forms the inferior boundary of the infra-clavicular region, and which, being continued behind, crosses the inferior angles of the scapulæ and seventh dorsal vertebra. It extends below as far as the twelfth rib, corresponding to the transverse line, carried round, which formed the lower boundary of the *infra-mammary* region in front.

As low as the eleventh rib lie the lungs. On the right side,

from the level of the eleventh rib, extending downwards, is the liver. On the left is the spleen, occupying some of the outer portion of the region; while the intestines occupy some of the inner part of the region. Close to the spine, on the left side, is the descending aorta; and on both sides, close to the spine, is a small portion of kidney (figs. 7 and 9, No. 14).

(B.) *Situation of the Organs in the Thorax.*

The *outer boundary* of each lung is marked by a line passing obliquely downwards and outwards from a little outside the centre of the clavicle towards the axilla, and then vertically at a variable distance outside the nipple. Each lung rises from half an inch to an inch and a half above the clavicle, the relative height being unequal but variable. The inner margin of each lung passes downwards and inwards from the apex, and meets with the inner margin of the other lung at the middle line, at a point between the first and second cartilages, or at the second.

The inner margin of the right lung continues vertically downwards along the centre of the sternum, or inclining a little to the left side, as far as the attachment of the xyphoid cartilage (fig. 10).

The inner margin of the left lung leaves the right at a point between the *fourth cartilages*, or a little higher or lower than this, and passes nearly transversely outwards for a short distance in the direction of the *fourth costal cartilage*. It then runs obliquely downwards and backwards in the course of a line drawn from the centre of the *fourth costal cartilage*, half an inch to one inch inside the left nipple, as low as the seventh rib (fig. 10).

The lower boundary of the right lung passes somewhat obliquely and then transversely from the attachment of the xyphoid to the sternum, across the cartilages of the sixth and seventh ribs backwards to the spine, which it touches on a level with the *tenth*, *eleventh*, or *twelfth* dorsal vertebræ (fig 9).

The lower boundary of the left lung is a little lower than that of the right, and passes backwards from the point indicated on the seventh rib, to strike on the spine at a point usually a little lower than that on the right side.

The apex of each lung lies beneath the anterior scalenus muscle and the subclavian artery. The apices of the lungs are separated

from each other by the œsophagus, the trachea, and the projection anteriorly of the bodies of the *first* and *second* dorsal vertebræ.

The base of the right lung is hollowed by the projection upwards

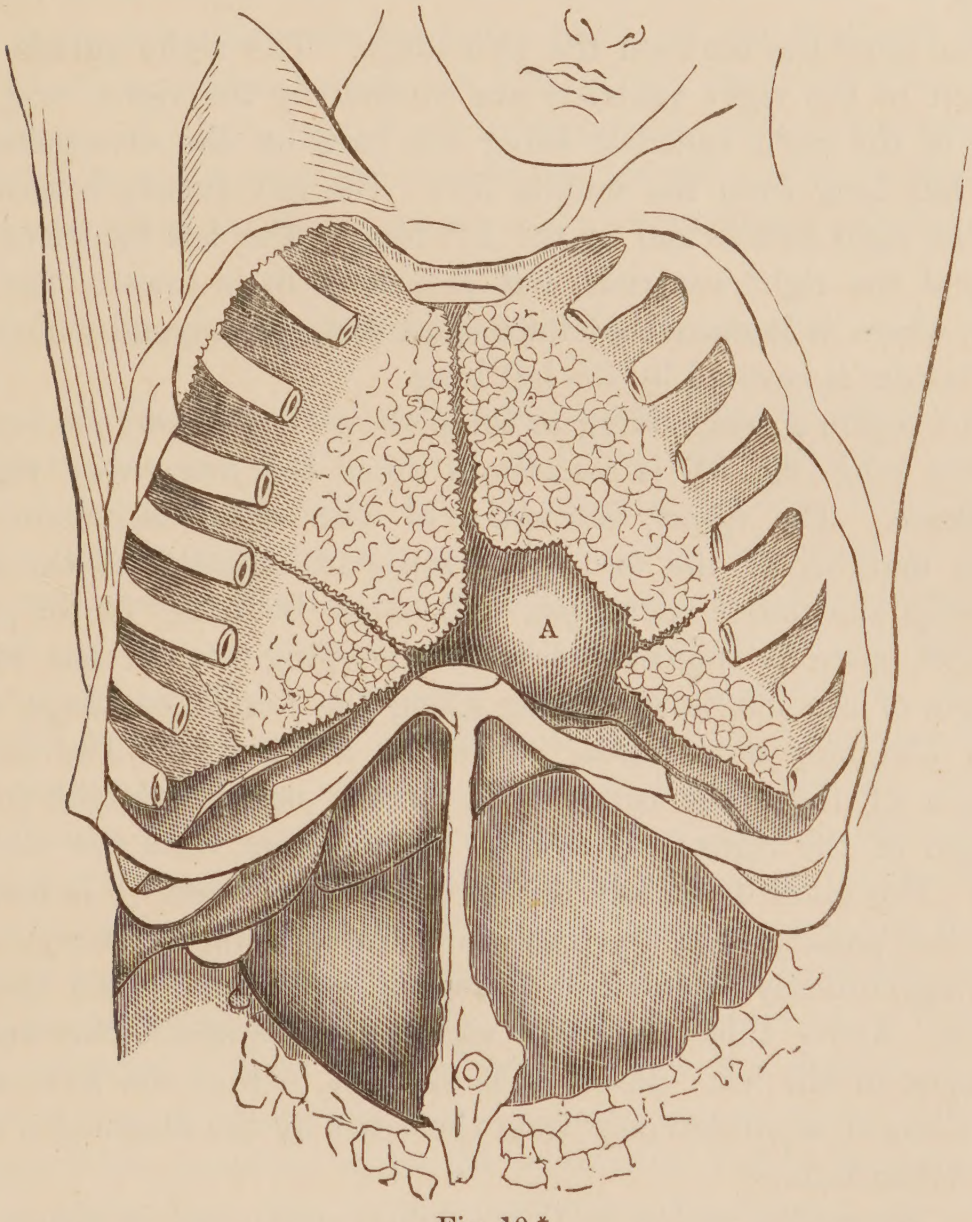

Fig. 10.*

of the liver, which in the centre of the thorax ascends as high as the *fifth* rib or *fourth* interspace. The liver is also separated from the ribs by the expansion of the lungs between it and the thoracic walls.

The base of the left lung may be also pressed upon by the left

* Relative positions of the margins of the lungs to each other, to the thoracic walls, and to A, the *præcordial* region, comprehending the right ventricle of the heart, covered by its pericardium (after Dr. Fuller. See his work *On Diseases of the Lungs and Heart*).

lobe of the liver; and it is always hollowed out, though to a less degree than the right lung, for the accommodation of the stomach and spleen, and, to some extent also, the left lobe of the liver.

The *heart* lies between the two lungs. The right auricle and a part of the right ventricle are covered by the right lung, the rest of the right ventricle being left bare by the divergence of the left lung from the middle line. The left auricle is covered by the right auricle and by the left lung. The left ventricle lies behind the right ventricle, but projects a little towards the left side, where it is uncovered for a short distance, beyond which its left border is covered by the left lung.

The region corresponding to the portion of the heart uncovered by the lung (fig. 10) is sometimes called the *præcordial* region or space. The *upper boundary* of this space is where the inner margins of the two lungs separate—namely, at the spot between the *fourth cartilages.* The *outer* boundary of the *præcordial* space is indicated by the diverging line of the inner margin of the left lung passing along the *fourth cartilage,* and then obliquely downwards, inside the left nipple. The *inner* margin of the space corresponds to the nearly straight inner margin of the right lung, behind the sternum, near the middle line. The *lower* boundary of the *præcordial* space is indicated by a line passing from the junction of the sternum to the xyphoid cartilage, directly to the left, or with a slight inclination downwards. Above this line is the right ventricle, and farther out is the apex of the left. Below it is the left lobe of the liver, and the stomach, separated only from the heart by the diaphragm and the pericardium.

The *præcordial* region is thus slightly pyramidal in shape, its base being about two and a half inches long, and nearly horizontal. Its inner margin is about two inches long, and nearly straight; its outer margin is from three to three and half inches long, and has a sloping direction from the apex to the pyramid (at the middle line between the *fourth* cartilages), to the outer extremity of the line indicating the base.

This *præcordial space* corresponds to the left half of the lower portion of the sternum, and to portions of the cartilages of the *fifth* and *sixth* ribs; and it may reach even to the junction of the cartilages with their ribs. Its inner and outer boundaries can be

marked out only by light percussion; and the lower boundary can only be defined with difficulty by the same means.

Below the boundary of the right lung the liver extends to the margins of the right false ribs, or a little below them. On the left side the space between the lower border of the lung and the false ribs is occupied by the left lobe of the liver, the stomach, the spleen, and by a portion of intestine—the colon principally. The left lobe of the liver stretches across beneath the xyphoid cartilage, and below this to a variable extent towards the left side.

(C.) *Changes in the Position of the Lungs.*

1. *In Health.*—(*a.*) During inspiration the lungs enlarge in all directions; the apices rise higher and the bases descend lower down; the points of union and of division between the inner margins are in the one case raised towards the apices, in the other case lowered down. The *præcordial* space is thus lessened in size by the advance of the anterior margin of the lungs. (*b.*) During expiration the state of things is reversed. The lungs fall from each other; but the point of division between the inner margins may be raised to a level with the *third* rib. The area of the præcordial space is increased. (*c.*) The difference between extreme inspiration and extreme expiration is considerable. In extreme inspiration the inferior boundaries of the lung are often from an inch to an inch and a half lower than they are in extreme expiration. (*d.*) The action of the heart causes a slight difference. Each impulse presses aside the sloping inner margin of the left lung; but this is so instantaneous that it causes no appreciable alteration when the *præcordial* region is mapped out by percussion. (*e.*) During respiration the thorax enlarges in all directions by the movements outwards and upwards of the superior ribs and sternum, and by the movements downwards and outwards of the inferior ribs. *In women* the movements of the upper ribs are much greater than in men, while the abdominal movements are less. The difference is increased by the use of stays; but it does not appear to be altogether owing to these. In boys the costal movements are often considerable: in old age they are diminished.

2. *Changes by Age.*—In children the still considerably developed thymus gland separates the inner border of the lungs at

the top of the sternum; and the point where they come in contact (converging from the apex) is lower than in adults. In children the lungs are also comparatively longer than in adults, and the inferior boundaries are lower down. In old people the lungs often alter considerably in shape, and produce corresponding alterations in position; the lower lobe, particularly in the left lung, becomes more posterior, and the upper lobes or lobe anterior. The lungs at last in old people become even larger above than below; and when mapped out by percussion, their several boundaries are found to have no certain and constant direction.

3. *Changes by Disease.*—One lung being incapacitated, the other lung undergoes supplementary enlargement; the inferior boundary is lowered, its inner margin is pushed to a variable extent across the median line over the heart, diminishing the *præcordial* space from the right or left side, according as the right or left lung is affected. So, also, if one lobe be affected, as by *pneumonia, tubercle,* or *cancer,* the other lobe, either upper or lower, as the case may be, is enlarged, and changes its position. In some diseases, as in emphysema, the lung enlarges: and if the emphysema is general, the lungs may meet each other almost at the top of the sternum; may not separate till on a level with the *sixth* rib; may leave the inferior margins at the *seventh,* or even the *eighth* rib; and may give a pulmonary percussion note in the posterior lumbar regions, below the ribs altogether. In cases of enlarged heart, or distended pericardium (unless there be coincident emphysema, or unless the lungs are floated forwards by *hydrothorax*), the lungs anteriorly are pushed aside, the point of separation of the inner margin is raised (especially in pericardial effusion), the inner margin of the right lung is thrown to the right side, and the inner margin of the left lung is thrown to the left side. Aneurism of the arch of the aorta, or tumors in the mediastinum, displace the upper portions of the lung; and tumors may even thrust aside and displace the heart. Abdominal diseases, as hepatic and splenic affections, peritoneal effusions, ovarian, uterine, or other tumors, also press up the thoracic organs, and alter their position. By such morbid states the inferior borders of the lungs may be not lower than the *third* intercostal space, or the *fourth* rib; the heart may be thrust upwards and outwards above and outside the left nipple. The student may also be prepared to meet with cases of more or less complete transposi-

tion of the viscera. (See a paper on this subject by Professor Allen Thomson, in the *Glasgow Medical Journal*, vol. i.; also in the *Lancet* for August 8, 1863, by my colleague, Professor Maclean.)

Section II.—Signs of Disease from the Shape of the Thorax.

The two halves of the thorax are seldom perfectly symmetrical. The right side in the most healthy persons often measures from *half an inch to an inch* MORE than the left; the right *infra*-clavicular space, particularly in right-handed persons engaged in laborious occupations, is apt to be slightly more prominent than the left; the *fourth*, *fifth*, and *sixth* left cartilages often project more than the right; the *infra*-mammary and *infra*-axillary regions may be larger on the left than on the right side; the *infra*-scapular region, on the contrary, is usually larger on the right side, or it may be markedly prominent on both. The nipples (which in adult males are above the upper margin of the *fourth* rib, near its junction with the cartilages) are always equidistant from the middle line, but the left is somewhat lower than the right.

Any marked changes beyond those now noted indicate disease either of the spine, as in *curvature;* or of the spine and ribs, as in *rickets;* or of the organs in the thorax. If *spinal curvature* and *rickets* are determined not to be present, any general expansions of one side as compared to the other, or general contraction, or (what is much more common) local expansion or bulging, or local contraction, depression, or excavation, become very important signs.

The existence of such conditions is ascertained by the eye—namely, by "*inspection*," and accurately by measurement with calipers or with tapes, or ingeniously devised instruments which have been named "*stethometers*" or "*chest measurers*."

General expansion of the thorax is usually produced by effusions of fluid into the pleura, or by extreme cancerous infiltration, or by emphysematous lungs. Much more rarely chronic consolidation will cause general or partial *bulging;* while tubercular deposition at its earliest period has been said also to cause some degree of local prominence; but *retraction* usually follows more or less complete impairment of the functions of the lung, or of a part of it, as in softened tubercle or cancer, and in the period of absorption of pleuritic fluid which has firmly compressed the

lung. Heart diseases give rise to local bulging only occasionally in the cardiac region. Aneurisms of the aorta may cause bulgings, while tumors may produce both bulgings and retractions according to circumstances (*MS. Notes* of Dr. Parkes' Clinical Course at University College).

Section III.—Physical Examination of the Chest.

I. **By Simple Inspection of the Form of the Thorax.**—It is necessary to note,—

(*a.*) Its general shape; and especially as to the condition of the *supra* and *infra*-clavicular spaces in respect of flatness, fulness, retraction, or bulging in these regions; the condition of the hollow above the notch of the sternum (the supra-sternal space); the form of the clavicles and their curvature; the height and breadth of the shoulders; the form of the sternum, as to whether or not it bends outwards or inwards; the curves of the spine; the position of the scapulæ; prominence of their inferior angles, and the firmness or laxity of the *latissimus dorsi* muscle; the distance of the scapulæ from each other and from the middle line; contracted contour or expanded form of the lateral regions; the width, depression, or bulging of the intercostal spaces; and distance of the nipples from the middle line.

(*b.*) Obtain a *general notion as to capacity or size of the thorax, relative to the height, weight, and age of the individual, allowing for fatness or emaciation,* (spirometry; see previous pages, under "*Scrofula,*" p. 190, vol. ii.)

(*c.*) Observe the thoracic movements, and estimate in seconds the time taken to complete the inspiratory and expiratory acts; compare the movements of the two sides of the thorax, and also the abdominal respiratory movements with those of the thorax, so as to notice if either takes an undue share in the work of respiration. If the ribs scarcely move, and the parietes generally of the thorax remain at rest, while the surface of the belly rises and falls alternately with the respirations, the act is called *abdominal respiration,* because the abdominal muscles seem to take the larger share in its performance; but if, on the other hand, no motion of the abdomen is visible, the act of respiration is then said to be *thoracic.* Observe whether the whole acts of respiration are quicker or slower than natural—*i. e.,* more

or less than *eighteen* to *twenty* per minute, or one to every four arterial beats. Note their frequency per minute. Observe whether they are calm, easy, and fully drawn, or short, forced, hurried, and incomplete, attended by indications of pain, checked or partially arrested by cough. Notice whether respiration is performed through the mouth or nose, or both; and whether the *nares* dilate and contract at each respiratory effort, with any constrained movement of forcible expansion. Note any sensible odour or vapour of the breath, and also its temperature.

II. **By Measurement.**—The use of tape or calipers in deep and medium inspiration and expiration will detect any differences between the two sides, or undue differences in the size of the chest at different times. There is about one inch of an average difference in favour of the right side of the chest compared with the left, and which is consistent with a normal state of the region. A convenient plan is the double tape, originally suggested by Dr. Hare. The double tape is formed by joining two common measuring tapes together, so that the beginning of each may be in the centre of the tape when joined. By putting the point of junction of the two tapes upon the spine, and holding it there tightly, the ends of the tape can be carried round the body at any point, and the circumference of each side read off simultaneously. By taking the size at full expiration and full inspiration, the extent of *expansion* is determined, as well as the absolute and relative size of each half of the thorax.

The measurements (besides the circular) which are most useful are the distance of the nipples from the middle line—their distance from the sterno-clavicular articulations of each side—and the distance of the centre of the clavicle from the lowest point of the false ribs in the vertical line.

The general expansion of a side of the chest is best learned by the use of the double tape, or by the use of some of the "*chest-measurers*" about to be noticed. The difference in the measurement between the fullest inspiration and the fullest expiration gives the general expansion of the lung. In health both lungs expand nearly equally from *three-quarters of an inch to an inch and a half;* or the right may expand a little more than the left. If there is any deficiency of expansion, there must be disease of some kind, but the nature of the disease cannot be known without additional signs.

Local expansion is most accurately determined by the eye, the

hand, and the chest-measurers of Drs. Quain, Sibson, or Leared; the exact levels of the measurements being always noted. All these instruments essentially consist of dials, with indices, moved by mechanism, connected with tapes passing round one or both sides of the chest—so many revolutions of the index indicating on the dial so many tenths and hundreds of an inch of expansion of the chest. Dr. Leared's instrument indicates differences on the two sides (*Med. Times and Gazette*, August 2, 1862); and Mr. Henry Thomson, of University College, has also suggested a simple addition to the tape measure, by which it is made differential. These instruments may be had of Coxeter, Weiss, Ferguson, Matthews, and other surgical instrument makers in London.

Dr. Sibson gives the following numbers as denoting the movements of various part of the thorax in health:—

(1.) The sternum and the first *seven* ribs in tranquil breathing advance forward from ·02 to ·07 inches. The left *fourth*, *fifth*, and *sixth* cartilages, and the *sixth* rib, move less than on the right side, on account of the position of the heart. In forced inspiration the movement forwards is from *half an inch* to *two inches*. (2.) The expansion of the *eighth* and *tenth* ribs varies from ·05 to ·1 inch. During deep respiration it is increased, but is less than that of the first five ribs. (3.) The abdomen moves forwards in tranquil inspiration from ·25 to ·3 inches. In deep inspiration the movement amounts to about *one inch*.

There is, however, very great variations in different persons; and "*chest-measurers*" must all be submitted (as *percussion* and *auscultation* are) to the test of comparison between the two sides —the difference which the heart causes between the two sides being remembered (Parkes, *MS. Notes*).

It is necessary in all these observations so to divert the patient's attention as to cause him to look away from the instruments, otherwise the movements of the chest may become so affected as to vitiate the results.

III. **By Palpation,** or the application of the hand. This method affords more extended information to that suggested by simple inspection, such as of the intercostal distances, lateral and antero-posterior; expansion of the chest in the acts of respiration; appreciation of *vibrations* communicated through the walls of the thorax. The palm of the hand applied to the chest in a healthy state during the act of speaking will appreciate a most delicate

vibratile tremor (commonly called *fremitus*), and more marked according to the graveness, coarseness, and loudness of the speaking voice. The intensity is generally greater in front than behind, on the right side than on the left, and is stronger towards the sternal than the humeral halves of the region below the clavicles. In disease the *fremitus* on the two sides of the chest must be compared. It is usually *increased* by whatever consolidates the fine vesicular texture of the lung, without obliterating the bronchial tubes. It is *diminished* by the intervention of liquid or air between the lung and the thoracic walls; also by such extensive consolidation of the lung-substance as to fill up the smaller bronchial ramifications leading to the air-vesicles. *Vocal fremitus,* or *vocal vibration,* is observed by placing the hand over the surface of the chest of a person speaking. It is a delicate vibration, easily deadened by the too forcible pressure of the hand. It is more marked in adults than in children, and in males than in females. It is more intense in long-chested than in short-chested persons; and markedly so in thin than in fat people; and is stronger in recumbency than in the sitting posture.

A *rhonchial fremitus* may sometimes be heard, when certain *rhonchi* throw the bronchial tubes into vibrations sufficiently strong to be felt on the surface of the chest. The sibilant, sonorous, and mucous rhonchi have all this property. *Friction fremitus* may be felt by the hand, when the gliding motion of the pleural surfaces upon each other is attended by a perceptible vibration, by the collision and friction of plastic matter upon the surfaces.

Ordinary fluctuation may sometimes be detected by the hands, combined with shaking or suddenly altering the position of the chest, as by *succussion.*

The hand is also used to appreciate the action of the heart. The heart, after full *expiration,* is felt to beat between the cartilages of the third and fifth ribs, and at the neighbouring part of the sternum, generally immediately below and to the outside of the left nipple. After a full inspiration it may be felt as low as the sixth rib. The frequency of the respirations may also be ascertained by the hand, applied to the surface below the clavicles in the female, and below the epigastrium in the male (WALSHE).

IV. **By Percussion.**—The position of the organs, as stated in the previous section, is ascertained during life principally by the

signs derived from percussion; that is, from the nature of the sound produced by striking over the lung either "immediately," as when the chest is gently tapped with the hand or a light hammer; or "mediately," as when a flat body is placed upon the chest, which is then struck by the finger or the hammer. The main object of percussion is to determine the comparative density of subjacent parts.

Mediate percussion is employed almost to the exclusion of immediate. The body which is interposed between the hammer and the chest is called a "*plexímeter*," and consists simply of a flat piece of ivory or wood, or a piece of india rubber, or of the index finger of the left hand laid flat upon the chest. The hammer is made of a piece of whalebone, or of light steel, armed at its head with a piece of india rubber; or the flat edge of the stethoscope encircled by a piece of india rubber; or it consists of a light thimble covered with vulcanized india rubber; or the points of the index finger, or middle fingers of the right hand, may be used for the purpose of a hammer, the nails being cut close.

Mode of Percussing.—The plexímeter is to be placed flat upon the chest, and held close to the walls. It is then to be struck perpendicularly, and with gentle or moderate force. If the fingers of the right hand be used as the hammer, the blow should be given from the wrist only, the elbow and shoulder being quite motionless. The force of the blow must vary according to circumstances. In the supra-clavicular spaces the finger, if it be used as a plexímeter, should be pressed closely into the triangular space, and the blow should fall in the direction of a line passing to the centre of the chest—namely, downwards, forwards, and inwards. If the blow be directed too much forward or backward, it strikes on the clavicle, or the parts at the back of the neck, and the "pulmonary percussion note" is not obtained. The clavicles are best percussed *immediately*, first at the sternal and then at the acromial end. In the *supra-spinous* spaces the blow should fall downwards, inwards, and forwards. Over the rest of the chest the plexímeter may be placed in any direction, provided the precaution is taken that corresponding points shall be struck on the two sides; as a slightly different note is given out, especially in thin people, by the intercostal spaces and the ribs. If the spaces are struck on one side, they should also be similarly, or with like force, struck on the other.

The "*pulmonary percussion note*" is the sound given forth by striking on a part of the chest-wall below which lies a considerable depth of lung. The sound produced does not depend upon the proper tissue of the lungs, but upon the vibrations of the air they contain, and also upon the vibrations of the walls of the chest. The pulmonary substance vibrates but little; it is non-sonorous, and acts rather as a damper of sound. The qualities of sound to be noted are—(1.) *Amount in intensity of resonance*, commonly called *clearness;* (2.) *Duration of the sound;* (3.) *Pitch;* (4.) *Volume of sound.*

The intensity of sound is diminished by the lessened vibration of the walls of the chest, as in pleuritic effusions; or by the lessened vibration of the air, the air being lessened in amount; or by increase in volume of the lung-substance, as by exudation.

The physical conditions which lessen the quantity of air are—(1.) Consolidation of lung-substance from any cause, such as exudation, tubercle, great congestion, or œdema; (2.) Compression of the lung from fluid in the pleura, or by tumors.

When the pulmonary percussion note is exaggerated, it may become what is commonly termed "*tympanitic;*" that is, a very clear sound, of less volume, but of higher pitch than the pulmonary percussion note. It is similar to the sound obtained by striking the abdomen when the intestines contain air, or a bladder moderately distended with air. It is generally caused by an increased quantity of air in the lung tissue or in the pleura, as in pulmonary emphysema, or when cavities exist in the lungs in pneumo-thorax. A very thin and flexible wall will often give a tympanitic character to the pulmonary percussion note. There are some exceptions to these statements, namely—(1.) When the air is not apparently in excess, a thin layer of sound lung full of air, lying over hepatized lung, will give forth the sound; (2.) The upper lobes of the lung when hepatized in the lower lobes will also give forth the sound; and (3.) When the lung floats on the surface of pleuritic fluid. *Tubular* sounds are produced when the lung is consolidated, covering bronchial tubes. In pleurisy the intensity of sound is diminished from both causes.

The sound is intensified or made more clear by whatever increases the vibrations of the walls of the chest, as in thin children, or in persons becoming thin, and by increased vibrations of air in the lungs.

V. **By Auscultation,** which implies "the act of listening," and is termed pulmonary or cardiac according as the sounds listened to relate to the lungs or to the heart. For this purpose the ear may be applied to the surface directly, merely interposing, for several obvious reasons, a fine towel or thin piece of linen or calico between the chest of the patient and the ear of the observer; or an instrument called the *stethoscope* may be used, and which is especially necessary when the sounds to be heard are limited to a small and definite region, as in listening to the sounds of the heart. The ear-piece of the stethoscope ought to fit the ear of the observer; or the ear-piece may be removed, so that the end of the stethoscope may be introduced into the orifice of the ear. It is important that the ear be well fitted. The extremity applied to the patient should be about an inch and a quarter in diameter.

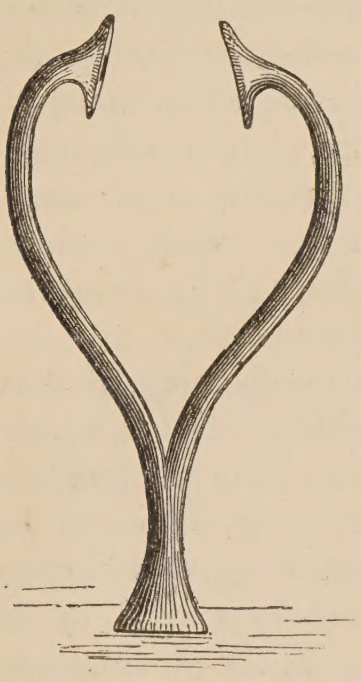

Fig. 11. *

A double or bin-aural stethoscope is also an instrument of great value in the differential diagnosis of sounds associated with lesions in the thoracic organs, especially when the sounds are obscure from their lowness. One form of the instrument seems to be an efficient magnifier of low sounds (LEARED), and another form of the instrument—that of Dr. Scott Alison—is extremely useful in cardiac diagnosis when the action of the heart is rapid or irregular (GAIRDNER). When the first sound is indistinct at the apex, or cannot be identified with the apex beat, or when the second sound is indistinct, or when it is audible only at the base, the first sound being audible only at the apex, the difficulty of recognition of the two sounds is very considerable. It is in such circumstances that the double or bin-aural stethoscope of Dr. S. Alison is of great value, for it enables the sounds to be identified at the points where they can be heard best; and it brings them into accurate relations with each other, because of the two ears receiving at the same time the sounds from two points of the *præcordial* region (GAIRDNER).

Dr. Arthur Leared, of London, devised and exhibited a double

* The Original Double Stethoscope of Dr. Leared.

stethoscope in the Great Exhibition of 1851 (Class X., No. 620), where it attracted little attention by the profession in this country. It consists (fig. 11) of two tubes, one for each ear, made of *gutta percha*, the elasticity of which, acting as a spring, keeps the tubes firmly applied to each ear. The thoracic ends of the tubes are fitted into a hollow cylinder or cup, which is applied to the surface of the chest. Dr. James E. Pollock, of London, called the attention of the profession to an American double stethoscope in the *Lancet* of April 12, 1855, at the request of his friend Dr. Coulson, neither of whom were aware that Dr. Leared had devised and exhibited such an instrument in 1851.*

Another form of double stethoscope has been devised by Dr.

* From discussions which followed in the medical journals, I have been led to make a statement in previous editions which does an injustice to Dr. Cammann; and I am glad to be able to insert this note from the American reprint of this text-book. The Editor of the reprint writes as follows :—

"The writer (Dr. Clymer) is indebted to Dr. James R. Leaming, of New York, the literary executor of the late Dr. Cammann, and his clinical assistant at the time named, for the following facts :—In the spring of 1852, Dr. H. W. Browne brought one of Marsh's patent double stethoscopes to Dr. Cammann's class at the Northern Dispensary. Dr. C. at once said that the principle was not a new one, and that he had at the time one of Landouzy's poly-stethoscopes sent to him from Paris, which was on the bin-aural principle. He also observed that it was a shame for a medical man to patent an instrument intended for the profession. He then proposed to his assistants to begin a series of experiments for the purpose of perfecting a stethoscope which would render the bin-aural principle practically available. With the assistance of Drs. H. W. Browne and C. P. Tucker, and the mechanic employed, the instrument known as 'the self-adjusting bin-aural stethoscope' was in the summer of 1852 perfected, and freely given to the profession without annoying restrictions. Soon afterwards he received a letter from Dr. Marsh, threatening a prosecution for the infringement of his patent. He took this letter to his lawyer, A. P. Mann, Esq., and authorized him to answer it, denying Marsh's claim to the discovery of the bin-aural principle; and, as the two instruments were widely different in construction, refusing to make any terms or compromise, and expressed

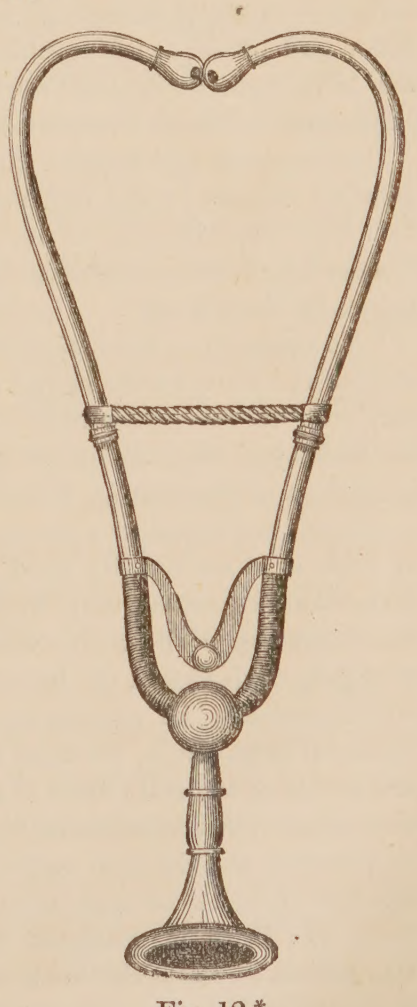

Fig. 12.*

* Self-adjusting Bin-aural Stethoscope of Dr. Cammann.

Scott Alison, and named by him the "differential stethoscope." Like Dr. Leared's instrument, it has a tube for each ear; but each tube has a separate cylinder or cup, which admits of being applied to any part of the chest. Dr. Leared's instrument enables us to hear sounds emanating from any given portion of the chest with both ears simultaneously; while Dr. S. Alison's instrument enables us to listen to the sounds emanating from two different parts of the chest at the same moment, so that we may compare the sounds at any two points, or on opposite sides of the chest, by a series of consecutive observations. If two sounds exist, one at each point, and if they differ in intensity, the weaker sound is eclipsed or nullified.

All forms of stethoscope require much practical study and

a willingness to contest the case at law if necessary. Thus not only did Dr. Cammann not patent the admirable mechanism of his stethoscope, which was clearly his own invention, but placed it freely in the hands of the profession, and stood ready to defend his right to do so. He had not been in Europe since 1830, and consequently could not have carried home Dr. Leared's idea (1851); nor is it probable he ever heard of Dr. Leared's stethoscope. He never called it 'Cammann's Double Stethoscope;' this was done by the instrument maker.

"It has been said that he did not appreciate the value of his own instrument, possibly because he did not assert his claim to its originality, and from the fact that after the first year of its introduction he used it sparingly, finding that its constant application to the ear sensibly dulled the delicate acuteness of hearing so prized by the expert.

"To understand the *rationale* of this effect, it is well to premise that the double stethoscope conveys the same sound impression through a distinct channel to each ear, just as the stereoscope presents an individual picture of the same object to each eye; and the effect in each case is to produce in the brain a clearer perception of the entirety of the sound heard or the object seen than can be done by one ear or one eye alone; but no instrument can do this perfectly unless both ears or both eyes are equally good. In ordinary sight or hearing, the eyes and ears being dual organs, one rests while the other is in action. Any one looking through the stereoscope a short time will be sensible of fatigue and temporary impairment of sight; and should it be too long and too constantly continued, the injury would probably be permanent; and the rule holds equally good in using the bin-aural stethoscope, for sight and hearing are cognate senses. It was a practical knowledge of the evil effects of a too constant application of the double stethoscope that caused Dr. Cammann to restrict its employment, believing it more important to preserve the delicate sensitiveness of the expert ear, than that the sound-impression should be made with its utmost power. It is gratifying to know that there are those in England who appreciate Dr. Cammann's unselfish labour. Dr. Alison, in his work *On the Chest*, pp. 322, 323, and 324, gives a description of this instrument, accompanied by a wood-cut, and accords to Dr. Cammann the full credit of rendering the bin-aural principle available. In describing the construction of his own instrument—the differential stethoscope—he acknowledges his indebtedness to Dr. Cammann in adopting the mechanism of his instrument, which he pronounces beautiful."

experience to use them with success. The ear-piece should fit the ear exactly in all forms of stethoscope; "and therefore, when a well-made stethoscope has been selected, and an ear-piece chosen which fits the ear comfortably, the student should keep to that one, and familiarize himself with its use" (FULLER).

The main object of the stethoscope is to *circumscribe* and localize the sounds that we hear; the chest end of the instrument should therefore be as small as possible, in order the better to appreciate the precise seat of the greatest intensity of sound. To ascertain this, the instrument should be "worked towards the sound," and then "from the sound," right and left, up and down, till its end is on the exact spot whence the sound proceeds in its greatest intensity. By working the stethoscope round and round, and gradually contracting the circle, the *area* of the sound can be ascertained; and hence so far the lesion producing it can be localized (HYDE SALTER).

Four circumstances affect the quality of a stethoscope:—(1.) *The material which allows the least amount of sound to be lost, and least of all perverts or modifies the sound,* is the best for a stethoscope—namely, some *porous* wood which is a good conductor of sound. Cedar and deal are the best woods for the purpose. The denser the wood the more are the sounds apt to be modified; therefore ebony is to be condemned. (2.) *The stethoscope should be of one piece of wood*—turned, in fact, out of a solid block. It ought not to be part ivory and part cedar. (3.) *As to length and thickness,* the length most convenient is that which permits the instrument to be carried in the crown of one's hat; and it may be solid if the wood is very porous; but generally it is hollow. (4.) *The chest end* should be small, not larger than *one inch and a quarter* in diameter; because the smaller it is, the greater is its localizing power, and the narrower are the limits within which the seat of any particular sound can be determined. The chest end of the stethoscope should also be narrow, and smoothly rounded over the edge. (5.) The *ear-piece* should be *large* and *flat,* to secure apposition and occlusion; but in this respect each man must fit his own ear (Hyde Salter "On the Stethoscope," *Brit. Med. Journal,* January 31, 1863). The wood-cut (fig. 13) represents the section of a good stethoscope as given by Dr. Hyde Salter. It is half the "natural" diameter—*i. e.,* one-eighth the size. The ear-piece is flat and broad, and the most careless application of the ear would produce perfect occlusion; the chest end

is small, with a narrow and rounded edge. The measurements are—length, seven inches; diameter of ear-piece, three; diameter of chest end, one and a quarter; circumference of shaft, one and a quarter.

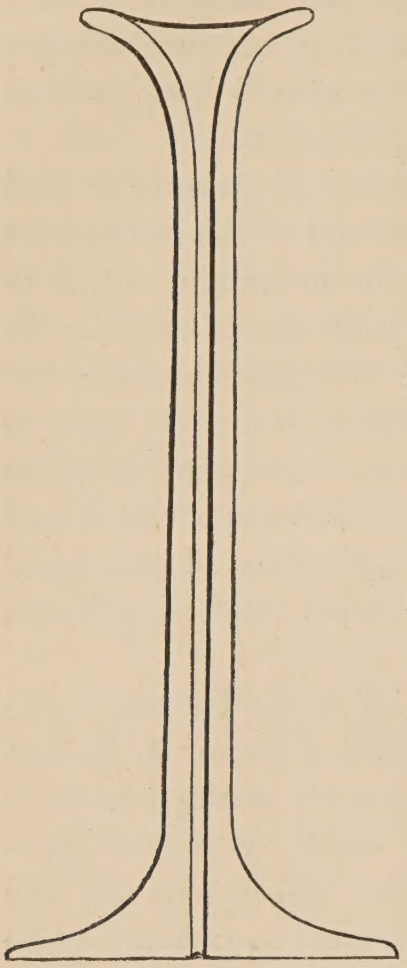

Fig. 13.*

[Dr. C. L. Hogeboom, of New York, has suggested an improvement in all stethoscopes, by stretching evenly and tensely over the pectoral extremity a piece of parchment, so as to be in contact with the skin, and sufficiently firm and elastic to compress the tissues and transmit sonorous vibrations. He claims that it intensifies the sounds, and that their source is more circumscribed. In Cammann's stethoscope it lessens the roaring. To auscultation of the larynx and trachea it is peculiarly adapted, as well as to that of the heart (*New York Med. Jour.*, vol. iii., 1866).]

To become familiar with the knowledge to be acquired by means of auscultation, much time and labour must be devoted to its practice, alike on persons in health and in disease. A verbal description of the sounds to be learned is difficult, because the impressions made on the senses of one person cannot be communicated exactly by language to another; and the distinctions which subsist between the sounds heard in health and those in disease are not yet regarded as similarly significant by all; nor is their individual importance yet clearly determined in relation to practice.

With the view of affording the means for a comparative study of the auscultatory phenomena, the following tables, relative to the natural sounds in health and the altered or morbid sounds in disease, are compiled from the writings of Walshe, Thompson, Wood, and Bennett; and it is hoped they may furnish an outline to guide the student in appreciating the morbid states of the lungs and heart.

The arrangement in a tabular form has been preferred, because it is believed the descriptions of the sounds are more easily studied and compared with each other by this mode of arrangement than any other.

* Section of a good form of Stethoscope (Hyde Salter).

TABLE I.—THORACIC *SOUNDS* OF *RESPIRATION* AND OF THE *VOICE* HEARD IN HEALTH.

Sounds of Respiration.	Synonym.	Character of the Sounds.	Common Site of Production.
Vesicular.	Respiratory murmur of inspiration and expiration.	*Inspiration.*—A soft diffused murmur of a gentle *breezy* character, increased in intensity with rapidity and force of respiration, and prolonged by a full inspiration. *Expiration.*—Slightly harsher and more hollow, weaker, and shorter, usually not above one-fourth the length of inspiration.	Entrance of the air and its expulsion from the air-cells and terminal portions of the bronchi—vibrations of tissue therewith connected.
Puerile.	Loud Vesicular murmur.	The respiratory murmur of children and women louder than that of adults, but with characters as above.	
Bronchial or Tubal.	Tracheal.	*Blowing* as of air passing quickly through a tube; higher in pitch than the vesicular sounds; more rapidly evolved. *Expiration* as long, or nearly so, as *inspiration;* generally a perceptible interval between *inspiration* and *expiration.*	Sites corresponding to the bifurcation of the trachea, the upper part of sternum, and between the scapulæ.
Sounds of the voice through the lungs. **Natural Bronchophony.**	Vocal resonance. Pectoral Vocal resonance.	Obscure, thrilling sound of the voice, diffused, and conveying the idea of distant origin. Articulation sometimes appreciable. Resonance more marked in males than females, and in adults than children; is only markedly present over the first bone of the sternum and in the inter-scapular regions.	The voice in articulation, passing down the trachea and bronchi, is obscured, intercepted, weakened, and diffused, by passing through the spongy pulmonary vesicular tissue to reach the surface of the chest.

TABLE II.—THORACIC *SOUNDS* OF A MORBID TYPE, SOMETIMES CALLED *RALES* BY FRENCH, AND *RHONCHI* OR *RATTLES* BY ENGLISH AUTHORS, EVOLVED DURING THE ACTS OF *RESPIRATION*.

A.—IN THE PULMONARY SUBSTANCE.

English Name of Sound (THOMSON).	Synonym.	Character of Sound conveyed by	Relation to Inspiration and Expiration.	How and Where Produced.	Diseases with which it is most usually Associated.
I. Bubbling.					
a. Bubbling rhonchi or rattles.	Mucous rhonchus or râle.	The bursting of bubbles of some size, unequal and varying in number, modified by coughing and expectoration.	Co-existing with both.	Bubbling of air through liquid (mucus, blood, or pus) in bronchial tubes of the size of a crow quill, and heard in the central or middle part of the lungs.	Bronchitis after secretion has become established.
b. Small bubbling rhonchus or rattle.	Subcrepitant or submucous râles or rhonchi.	The bursting of more minute bubbles, producing weaker sounds.	Co-existing with both movements, but predominating during inspiration.	Bubbling of air through more or less viscid fluid in minute bronchial tubes, as at their peripheral distribution.	Capillary bronchitis of both bases of lungs, tubercular bronchitis of apex, resolution of pneumonia.
c. Gurgling rattles.	Cavernous rhonchi or râles.	The bursting of bubbles obviously of large size, with a hollow gurgling sound, or a metallic sound if the bubbles be small.	Co-existing with both acts.	Caused by the bursting of bubbles in a hollow space, enclosed by more or less dense and smooth walls—the more so, the more perfect the gurgling.	Excavations from tubercle or other causes, dilatations of bronchi, pus in the pleura, with a bronchial fistula.
II. Clicking, or Crackling.	Humid crackling, humid crepitation, humid crackling rhonchus.	Successive clicks, few in number, and tending to pass into the bubbling rhonchi, especially during expiration.	Co-existing with both acts, but more regular and distinct during inspiration.	Mechanism of its production obscure. It probably originates in the interior of softened tubercles, which have just commenced to communicate with the minute bronchi.	Generally in *direct* connection with tuberculous exudation which has commenced to soften.
	Dry crackling rhonchus, dry crepitation.	A succession of three or four minute dry, short, sharp, crackling sounds, permanent in many cases when once established, and tending to pass into the clicking sound. At first it may disappear for a day or two and again recur.	Co-existing exclusively *with inspiration.*	Mechanism of their production undetermined. Conveys the impression of being evolved at a distance from the surface, and in the great majority of cases is found in the *infra-clavicular* and *supra-clavicular* regions.	Generally observed on the eve of the softening process in tubercles.

III. Crepitation. *a. Primary.*	Crepitant râle or rhonchus.	The idea of crepitation, like that produced by rubbing slowly and firmly between the finger and thumb a lock of one's hair near the ear (WILLIAMS). An immense number of sharp sounds, conveying the notion of minute size and dryness.	Co-existing exclusively with *inspiration*, and at first towards its close only.	Probably due to the sudden and forcible expansion of delicate tissue, altered in its physical properties by the inflammatory state, and which probably undergoes minute ruptures.	Primary idiopathic pneumonia, or the pneumonic state established round tubercles. The crepitation of early pneumonia.
b. Secondary.	*Rhonchus crepitans redux.*	Crepiti of a bubbling nature, slowly evolved, few in number, and unequal or dissimilar and irregular in occurrence.	Audible in expiration as well as inspiration, but pertaining specially to the latter.	Probably due to the bubbling of air through fluid contained in the minute bronchi.	Co-existing with the resolution of pneumonia.
IV. Vibration. *a. Sonorous rhonchus.*	Sonorous râle.	A musical sound of a vibratory, deep, or grave tone, attended with fremitus of the walls of the thorax over a variable surface. *Snoring*, *humming*, *cooing*, and *bass* notes are its varieties.	Co-exists with inspiration and expiration, but especially marked in the latter, to which it may be limited.	Arises in the larger bronchi, and suggests the idea of vibrations.	Essentially associated with bronchitis.
b. Sibilant rhonchus.	Sibilant râle.	A high-pitched whistling sound, of variable intensity and duration, and irregular recurrence. *Clicking*, *whistling*, and *hissing* varieties are described.	Co-existent with inspiration and expiration, especially marked in the former, but occasionally limited to either.	The influence of the passage of air on a local accumulation of viscid mucus.	Pulmonary emphysema and bronchitis.
B.—ASSOCIATED WITH THE MOTIONS OF THE PLEURA.					
V. Friction.	Grazing, rubbing, grating, creaking, are varieties of this sound.	The sensation of friction by a series of abrupt jerking sounds, rhythmical with respiration, few in number and superficial in seat, limited in extent, attended with fremitus, palpable to the hand, and perceptible to the patient.	Invariably heard in inspiration, or in both respiratory acts.	The rubbing of two opposed serous surfaces together, attended by inflammation.	Pleurisy, or any cause of roughness on the surface of the pleura.

TABLE III.—THORACIC *SOUNDS* OF A MORBID TYPE EVOLVED DURING THE ACT OF *ARTICULATION* OF THE *VOICE*.

Name of the Sound.	Character of the Sound.	Physical Conditions under which it is supposed to be Produced.	Diseases with which it is commonly Associated.
I. Bronchophony.	Exaggerated resonance of the voice, unattended with articulation, diffuse or concentrated, and rarely producing any tactile sensation to the ear.	Increased density of the pulmonary tissue surrounding pervious bronchi, with enlarged calibre and hypertrophy of the substance of the bronchi. The more homogeneous the consolidation the better probably is the sound transmitted.	Hepatization of lung; dilated bronchi; pleurisy, with effusion, when hepatization co-exists.
II. Pectoriloquy, or Pectoriloquous Bronchophony.	Complete transmission of articulated words from the walls of the chest into the ear; the resonance being generally circumscribed and limited in extent.	When solid masses of lung lie between a large bronchus and the parietes; when a moderate-sized excavation exists, with smooth and dense internal surface.	Various morbid states in which such physical conditions may exist.
III. Amphoric resonance.	A ringing metallic sound reverberating through a cavity, and resembling that produced by speaking into a broad-mouthed empty pitcher.	The voice reverberating in a large cavity, communicating with a bronchus by a small aperture.	Chiefly in phthisis.
IV. Ægophony.	A sound—vibratory, tremulous, cracked, and irregular, limited in its seat, comparable to the bleating of a goat, or to the voice of the exhibitors of Punch; synchronous with the articulation of each word, or following like a feebly whispered echo from a distant source. The sound appears to flutter tremulously about the applied end of the stethoscope.	When a stratum of fluid contained in the pleura compresses the lung, the voice in the bronchial tubes is thus rendered more distinct, by the compression of the pulmonary texture, and is thrown into vibration by the layer of fluid.	A tendency to it during the early period of pleuritic effusion, equally diffused and small in amount.

Section IV.—Relation of the Parts of the Heart and great Blood-Vessels to the Walls of the Thorax.

A knowledge of the exact position of the several parts of the heart, particularly of its valves and orifices, and of their relation to fixed points on the surface of the chest, is essential to accurate diagnosis. The size of the heart, and of its several parts, may thus be relatively determined in the living subject: so also its relative position, and any amount of displacement it may have undergone, and whether or not its valves or orifices are diseased. The base of the heart being the most fixed part, is the most convenient from which to trace the outline of the heart, and to determine what parts correspond to certain fixed points upon the surface of the chest.

The heart is situated obliquely in the cavity of the thorax, from above downwards, from before backwards, and from right to left. It lies behind the middle and lower bone of the sternum, also behind the cartilages of the third, fourth, and fifth right ribs, near the sternum, and the cartilages of the third, fourth, fifth, and sixth ribs on the left side, in front of the bodies of the sixth, seventh, and eighth dorsal vertebræ. It rests immediately above the diaphragm upon its cordiform tendon, the serous layer of the pericardium only being interposed. Owing to the obliquity of its position, the *line of the base of the heart* looks upwards and backwards towards the right shoulder. *The apex* points downwards and forwards towards the space between the cartilages of the fifth and sixth ribs on the left side, where its impulse may be felt during life. *The base of the heart* is on a line with the interval between the cartilages of the second and third ribs.

The region of the heart's superficial dulness is known as the *præcordial* region, the limits of which, as already defined, correspond to a vertical line through the centre of the sternum; and about the middle of the bone, nearly on a line with the cartilage of the fourth rib, the edge of the left lung separates from this middle line, and passes obliquely to the left side—thus exposing a small portion of the pericardium (figs. 5, 6, *ante*), which is uncovered by lung. The exposed surface has a triangular shape; the apex above, the base below. The parts of the heart thus exposed beneath the pericardium are a part of the left ventricle, near its

apex, and a portion of the apex of the right ventricle. This triangular *præcordial* space is on a plane below the nipple and the fourth rib. Its base is on a line with the cartilage of the sixth rib; its right boundary nearly a vertical line through the centre of the sternum; its left boundary is an oblique line through the cartilages of the fifth and sixth ribs on the left side. Within these limits the heart is in contact with the parietes of the chest, yielding a characteristic sound to percussion.

The sac which encloses the heart has a pyriform shape, the base below, the apex above, exactly the reverse of that of the heart. Thus, the *base of the pericardium* is on a line with the upper part of the xyphoid cartilage; *its apex* is a short distance above the origin of the large vessels, and generally on a line with the articulations of the cartilage of the second ribs with the sternum; but may extend as high as the level of the articulation of the first ribs with the sternum. The sac is wider at the centre (corresponding to the greatest transverse diameter of the heart) than it is at the base; and towards its centre it extends more towards the left side.

The line of the base of the ventricular portion of the heart is from three to three and a half inches below the clavicles (left and right respectively), and on a line with the junction of the cartilage of the third left rib and fourth right rib with the sternum.

The line of the base of the left ventricle rises as high as a line drawn across the junction of the cartilage of the *third left* rib with the sternum—*i. e.*, about three inches below the clavicle on that side.

The line of the base of the right ventricle corresponds to a line across the upper margin of the junction of the cartilage of the *fourth right* rib with the sternum—*i. e.*, about three inches and a half below the clavicle on that side.

The impulse of the apex of the organ is to be felt between the fifth and sixth left ribs, near where the body of these ribs joins the cartilage. The apex is a little below the fifth left rib, slightly to the left of its junction with its cartilage, and on a line with the articulation of the xyphoid cartilage with the sternum. The nipple in the male has been considered to be a useful guide. It is said to be upon the fourth rib, or over its upper or lower edge, a little more than an inch to the left of the junction of the rib with

its cartilage. The edge of the left ventricle reaches the nipple on the left side.

The length of the ventricular portion of the heart is determined by the length of a line drawn from the middle of the sternum, between the cartilages of the third ribs, to below the fifth left rib, slightly to the left of its junction with the cartilage.

The greater part of the *right ventricle* lies behind the sternum: at its upper part it extends slightly to the right of this bone, a small portion extending under the cartilages of the fourth and fifth right ribs close to the sternum. Its apex is to the left of the sternum, a little above the apex of the heart; and a part of the right ventricle extends under the cartilages of the fourth and fifth right ribs, close to the sternum. The inferior margin of the right ventricle is nearly on a line with the junction of the xyphoid cartilage and the sternum. The anterior wall lies immediately under the sternum. It is overlapped at its upper portion by both the right and the left lung.

The *left ventricle* is covered by the left lung; and all its anterior surface is to the left of the sternum, extending from the cartilage of the third left rib to the interspace between the fifth and sixth left ribs, near where the cartilage joins the body of these ribs. It lies between the sternum and the nipple on the left side, to which its left margin reaches.

The *right auricle* lies to the right of the sternum, entirely covered by the right lung. Its appendix lies behind the cartilage of the third right rib, its tip rests against the right side of the ascending portion of the arch of the aorta, and is on a line with the pulmonary valves.

The *left auricle* is entirely covered by the left lung. Its appendix is the only portion seen when the pericardium is laid open. It lies behind the cartilage of the third left rib, close to the sternum, resting against the left side of the commencement of the pulmonary artery.

The line of the base of the auricles is on a line with the interval between the junction of the second and third ribs with the sternum, the greater portion of it being under the sternum.

Relative Position of the Orifices of the Heart.—The *right auriculo-ventricular orifice* lies behind the centre of the sternum, on a line with the lower margin of the articulation of the cartilages of the fourth rib with the sternum.

The *left auriculo-ventricular orifice* is on the same level, but on a plane posterior to the right. It lies behind the cartilage of the fourth left rib, near to or behind the sternum.

The *valves of the pulmonary artery* are on a line with the space between the cartilages of the second and third ribs to the left of the sternum, and very close to this bone. In some instances they may lie a little lower down—namely, on a line with the junction of the cartilage of the third left rib with the sternum, and immediately under it.

The *aortic valves* lie behind the sternum, on a line with the junction of the cartilages of the third rib with the sternum, and towards the left edge of this bone. When the valves of the *pulmonary artery* are situated lower down, the *semilunar valves* of the aorta will be lower also, and on a line with the interval between the insertion of the cartilages of the third and fourth ribs.

A line drawn across the inferior margin of the third ribs corresponds to the *base of the valves of the pulmonary artery*, and to the *free border of the aortic valves.*

The *right ventricle* ascending higher than the left, *the orifice of the pulmonary artery* is on a plane higher than that of the aorta; hence the *pulmonary orifice* is the highest up, as well as the most anterior, of all the orifices of the heart.

The *aortic orifice* lies behind the pulmonary orifice, but on a lower plane.

The *left auriculo-ventricular orifice* is immediately behind the aortic orifice, but on a lower plane.

The *right auriculo-ventricular orifice* is nearly on the same plane as the left, but more anterior, about three-quarters of an inch lower than the pulmonary orifice.

The *ascending* portion of the arch of the aorta curves to the right of the sternum, between the cartilages of the second and third ribs. In this part of its course it is still within the pericardial sac, and (in the dead subject) lies at the depth of an inch and a half from the surface, the margin of the right lung and the pericardium being between it and the parietes of the chest.

The *transverse portion of the arch of the aorta* crosses the trachea at the centre of the first bone of the sternum on a line with the lower margin of the articulation of the cartilages of the

first ribs with the sternum, and at a still greater depth from the surface.

The *arch of the aorta* approaches most closely to the parietes of the chest at the point at which the *arteria innominata* comes off —i. e., *on a line with the junction of the cartilage of the second right rib with the sternum.*

The *origin of the pulmonary artery* is on a line with the junction of the cartilages of the third ribs with the sternum. The tip of the left auricle rests against its left side. The pulmonary artery ascends about two inches before it divides; and at that point a portion of the margin of the vessel comes to the left of the sternum between the cartilages of the second and third ribs. The division of the artery is on a line with the upper edge of the cartilage of the second ribs where they join the sternum.

The *ascending vena cava* passes through the diaphragm by an opening, which corresponds to the upper part of the xyphoid cartilage.

In various morbid states the apex of the heart is formed by the *right ventricle* alone, or by the right and left together: in hypertrophy of the right side, for example, associated with *bronchitis* or with *emphysema.* When the right ventricle chiefly enters into the formation of the apex of the heart, the apex is then broad and rounded, rather than of the normal conical form, and is evidence of long-standing pulmonary obstruction (BELLINGHAM, BOUILLAUD, FULLER, PEACOCK, WALSHE, WILKS).

SECTION V.—DIMENSIONS OF THE ORIFICES OF THE HEART.

The healthy working of the heart is consistent rather with its relative size, and the relative dimensions of the orifices to each other, to the size and weight of the body generally, the condition of other organs, such as the lungs, the liver, the stomach, the spleen, and the kidneys, than with the absolute size of the heart itself.

The *tricuspid orifice* ought to be larger than the *mitral orifice,* and *half as large again* as the *aortic orifice.*

The *mitral orifice* ought to be larger than the *orifice of the pulmonary artery,* and larger than the *orifice of the aorta* by *one-fourth.*

The *orifice of the pulmonary artery* ought to be larger than the orifice of the *aorta* by about *one-eighth* (WILKS).

The *mitral orifice* tends to diminish under all forms of disease which diminish the supply of blood coming from the lungs to the left side of the heart—*e. g.*, smallness of the chest and lungs compared with the size of the person, and imperfect respiratory acts. Such forms of contraction are independent of organic disease of the part, and are unlike induration, which implies increased development of tissue round the orifice which leads to its contraction. Under this latter condition ulceration may ensue, when the anterior curtain is generally partially destroyed, when large soft vegetations or concrete fibrin are deposited from the blood, and are found adhering to the diseased parts.

If the *mitral orifice* is relatively small and contracted, the left auricle may become surcharged with blood. Its walls are then apt to become thick, tough, and hypertrophied, in place of being uniformly thin. Pulmonary obstruction is associated with this condition, and enlargement of the right side is a subsequent effect.

Aortic obstruction or contraction enlarges relatively all other parts of the heart.

The orifices of the right side of the heart exceed in size those of the left, and more especially the *auriculo-ventricular orifices*, compared with the arterial orifices; but in advanced life the *aortic orifice* tends to exceed the *pulmonary*.

The researches of Bizot likewise show that the size of all the orifices of the heart is relatively greater in the male than in the female, and that their size progressively increases as life advances in both sexes.

The following are the average dimensions of the orifices of the heart given by Bizot:—

	Male.	Female.
Right auriculo-ventricular orifice, ...	54·12 lines.	48·25 lines.
Left auriculo-ventricular orifice, ...	45·17 ,,	41·3 ,,
Pulmonary orifice, ...	32·2 ,,	30·7 ,,
Aortic orifice, ...	31·15 ,,	28·4 ,,

Section VI.—Relative Weight of the Lungs and Heart.

The absolute weight of the lungs and of the heart increases as life advances, and is greater in the male than in the female at all ages (Bizot, Boyd, Peacock).

The following are the records given by Dr. Boyd in his paper already referred to:—

TABLE SHOWING THE RELATIVE AVERAGES OF BODY-WEIGHT, AND THE WEIGHT OF THE LUNGS AND HEART, AS TO AGE AND HEIGHT.

Age and Sex.	Body-weight.	Body-height.	Weight of Right Lung.	Weight of Left Lung.	Weight of Heart.
Years.	Lbs. Oz.	Inches.	Ounces.	Ounces.	Ounces.
1 to 2 {Male,.....	14 6	28·5	4·12	3·99	1·66
{Female,.....	13 2	27·7	3·58	2·95	1·47
2 to 4 {Male,.....	20 0	31·6	5·45	5·08	2·14
{Female,.....	18 7	31·6	5·04	4·24	2·11
4 to 7 {Male,.....	25 8	37·5	6·27	6·01	2·77
{Female,.....	24 9	37·0	5·52	5·45	2·3
7 to 14 {Male,.....	42 6	47·0	10·14	10·38	4·25
{Female,.....	38 6	45·0	8·82	8·17	4·38
14 to 20 {Male,.....	68 0	60·5	20·4	19·67	7·61
{Female,.....	63 14	57·7	17·52	15·08	8·46
20 to 30 {Male,.....	92 14	66·75	32·34	30·09	10·06
{Female,.....	86 13	62·0	21·89	16·71	9·08
30 to 40 {Male,.....	98 3	66·5	28·47	24·29	11·36
{Female,.....	87 0	62·0	18·74	17·64	9·45
40 to 50 {Male,.....	102 0	66·8	31·21	28·63	11·53
{Female,.....	84 9	62·0	19·73	17·47	9·6
50 to 60 {Male,.....	102 0	66·0	30·32	25·29	11·83
{Female,.....	86 0	62·0	19·48	17·08	10·44
60 to 70 {Male,.....	103 13	65·7	28·52	24·16	12·94
{Female,.....	86 14	61·5	20·32	16·59	10·64
70 to 80 {Male,.....	106 13	65·7	18·77	17·11	13·14
{Female,.....	80 4	61·0	16·76	14·59	10·10
Above 80 {Male,.....	99 0	66·7	30·46	24·30	12·1
{Female,.....	79 5	60·0	18·22	15·23	10·27

The thickness of the parietes of the heart varies also with age; those of the *left* ventricle are thicker in the male than in the female at every age, and the thickness increases as age advances. The parietes of the right side increase, but in a much less ratio.

The thickest part of the parietes of the left ventricle is at the centre, next at the base, and it is thinnest at the apex (the mean thickness is under *six* lines). The thickest part of the parietes of the right ventricle is at the base, where it is above *two* lines. The thickest part of the *septum ventriculorum* is at its centre.

Section VII.—Mode of Examination of the Heart.

In order to determine the nature, the situation, and extent of morbid changes in the heart, it is necessary to be able to recognize readily any alteration in the heart's impulse, either as regards its strength or the situation in which it is felt—it is necessary to be able to detect any difference in the extent and degree of the heart's superficial dulness, or any change in the character of its sounds different from those which are normal.

Inspection, palpation, percussion, and auscultation are all therefore capable of affording valuable assistance in arriving at a diagnosis; and the following statements are condensed from the admirable writings of Drs. Bellingham, Fuller, Gairdner, Stokes, and Walshe.

The inspection of the external surface of the thorax, and the application of the hand to the *præcordial* region should never be omitted. Positive information is obtained by these two methods of examination, which mutually assist one another.

The exact point at which the apex of the heart comes in contact with the parietes of the chest may be determined simply by inspection. The *strength* or *feebleness* of the impulse of the heart is to be determined by the application of the hand.

By inspection it is ascertained whether the two sides of the thorax are symmetrical; and (in connection with cardiac diagnosis) whether there is any bulging in the *præcordial* region, or any unusual pulsation at any part of its parietes in the large arteries which come off from the arch of the aorta, as well as in the jugular veins or epigastric region.

By *the application of the hand*—palpation—the force or impulse of pulsation is determined, the frequency or slowness of the heart's action is judged of, and the regularity or irregularity of its movements. We may likewise appreciate by this means any *tremors* or *frictions* which accompany its action in the pericardium. To determine the impulse of the heart, the hand must be placed directly upon the surface of the chest; but *mediate* palpation may be used by placing one end of the stethoscope over the part where the impulse is, when the extent to which the instrument is elevated, and the force with which this is accomplished, will give an accurate idea of the strength of the

heart's impulse, especially in hypertrophy, or in hypertrophy with dilatation of the ventricles.

In healthy persons with well-formed chests the impulse of the heart is so slight as not to be perceptible to the individual himself; and it is felt only at one spot—namely, between the cartilages of the fifth and sixth ribs on the left side—*i. e.*, from one to two inches below the nipple, and to its sternal side. When the parietes of the chest are much loaded with fat, the impulse is scarcely perceptible to the hand; while in thin persons it is evident to the eye.

The impulse is somewhat stronger in the erect than in the recumbent posture. A forced inspiration diminishes it, and causes it to be felt lower down than usual; while a deep inspiration elevates the ribs, without raising the heart in the same degree.

In a forced expiration the impulse is more perceptible, and is felt higher up. In examining the heart it is therefore necessary to make the patient vary his position, and to examine the heart both during inspiration and expiration. Calmness and tranquillity on the part of the patient must be obtained, because mental excitement, as well as exercise or exertion, increases the impulse of the heart. A diminished impulse, circumscribed or feeble, is due to feebleness of the action of the heart from disease or alteration of its muscular tissue—as in softening or fatty degeneration of its tissue, or general debility of the system; or owing to disease in the lungs or pericardium—as from effusion into the sac—when the apex of the organ may be prevented from coming in contact with the parietes of the chest. The impulse is also diminished in cases of attenuation of the walls of the ventricles, with dilatation of their cavity. Emphysematous lungs may likewise overlap the heart, and prevent its impulse from being felt. Increased impulse of the heart is generally due to some morbid state of the heart itself. It is stronger than natural in hypertrophy of the walls of the left ventricle, and is greatest in hypertrophy with dilatation of the ventricles. In such cases the impulse is slow, gradual, heaving, double, and occasionally so violent as to shake the bed on which the patient rests. This slow, progressive, heaving impulse is produced by no other cause than hypertrophy with dilatation of the ventricles of the heart; and in such cases the extent of surface over which the impulse is felt is much increased, and the whole side of the chest is sometimes

elevated by the action of the organ. The double impulse which can be felt is due to the fact that the *diastole* as well as the *systole* of the ventricles is accompanied by an appreciable impulse. Adhesions of the pericardium to the pleuræ of opposite sides may so bind down an enormously large heart that its impulse will not be felt.

The situation of the impulse of the heart may be altered by displacement of the heart itself, as in cases of empyema, towards the left side when the right pleural cavity is distended with fluid, and to the right side when the left pleural cavity is distended with fluid. The impulse may then be felt on the right of the sternum. In cases of ascites the heart may be pushed upwards, and its impulse felt on a plane higher than natural; so also in cases of ovarian and other abdominal tumors, hysterical tympanitis, or in advanced stages of pregnancy. In emphysema of both lungs the heart is displaced downwards, so that the impulse is then felt sometimes as low down as the epigastric region. When fluid is effused into the pericardium, the site of the impulse is somewhat elevated; and as the amount of fluid increases, the impulse becomes weaker, unequal, undulatory, or irregular; and when the effusion is very considerable the impulse will be altogether absent.

In hypertrophy of the left ventricle, with dilatation of its cavity, the impulse is felt lower down than natural, more to the left side, and occasionally on a line vertically from the axilla.

In hypertrophy with dilatation of the right ventricle, the impulse is felt lower down, and more to the right side than natural, and not unfrequently on a line with the xyphoid cartilage.

In the former case (hypertrophy with dilatation of the *left* ventricle) the impulse is progressive, heaving, and strong, elevating the hand or stethoscope of the observer, and felt over a very much larger surface than natural; in the latter case (hypertrophy with dilatation of the *right* ventricle) the impulse is felt over a more circumscribed space, and is neither heaving, prolonged, nor very strong. But the downward displacement of the heart is not due *only* to the hypertrophy. Three causes conspire to produce it: one is the hypertrophy of the organ; another is the dyspnœal inflation of the lungs; and another is the flattening of the diaphragm, which always exists where there is an abiding

source of dyspnœa. This descent and flattening of the diaphragm is due to the instinctive efforts made in all forms of dyspnœa to obtain more air. The large heart is thus pushed downwards by the dyspnœal lung inflation, and pulled downwards by the diaphragm (Hyde Salter, *Brit. Medical Journal*, February 8, 1862).

Turgescence and pulsations in the jugular veins are to be appreciated by inspection and palpation. They are signs which accompany advanced stages of some cardiac diseases. Turgescence is the most common, and occurs in cases in which an impediment exists to the free passage of the blood through the right side of the heart; and in such cases, when the tricuspid valve imperfectly closes the right auriculo-ventricular orifice, and so permits regurgitation into the auricle at each systole of the right ventricle, we have pulsation of the jugular veins as well as turgescence. Pulsation is always most evident immediately above the clavicles, and may extend half-way up the neck.

When the *mitral valve* permits regurgitation, a "purring tremor" may be felt when the hand is placed over the region of the *mitral valve;* and a similar tremor may be felt over a dilated aorta when the valves are patent, or when a communication exists between a vein and an artery. The jarring pulse in the radial artery, in cases of aortic valve patency, is an instance of the "purring tremor" felt in an artery of small calibre.

Percussion.—The whole of that portion of the anterior wall of the chest behind which the heart is situated is sometimes called the *præcordial region;* but *anatomically* it is a region more limited, corresponding to that part of the pericardium of the heart uncovered by lungs (fig. 10, *ante*, p. 525).

Both regions, however, are definable by the limits of a dull sound on percussion and more slight percussion.

The portion of heart uncovered by lung seldom exceeds *two inches* in any direction. It has a triangular shape, the base below, the apex above. It consists of a portion of the apex of the right ventricle, and of part of the left ventricle near its apex, and is on a plane below the nipple and the *fourth* ribs. Its base is on a line with the cartilage of the sixth rib. Its apex is at the point where the margins of the opposite lung begin to separate from one another—*i. e.*, immediately below the *fourth* rib. The triangular boundaries of this anatomically true *præcordial space* are

constituted—(1.) On the right side of the thin edge of the right lung, by nearly a vertical line through the centre of the sternum; (2.) On the left side of the thin margin of the left lung, by a more or less oblique line through the cartilages of the *fifth* and *sixth* left ribs; (3.) Below, it is bounded by the diaphragm.

Over this region, to slight percussion, a sound less dull than that yielded by the liver is elicited, and on stronger percussion a difference of sound can be detected where the thin margin of the lungs covers the heart. The mode of percussing this region, so as to mark the lung part and the heart part, is to lay one finger over the decidedly dull part, and another over the slightly resonant edge of the lung, when, by striking the two fingers alternately, the arched line along which the organ lies in contact with the walls of the chest may be traced with accuracy, unless fat obscures the resonance (HOPE).

Another means of estimating the size of the heart is by *auscultatory percussion.* This requires two competent persons to determine the result, and is managed as follows:—"A solid cedar cylinder, six inches in length and one inch in diameter, cut in the direction of the fibres, and with an ear-piece attached, is applied to the centre of the *præcordial* region, while the ear is applied to the other end: percussion is then made by another person from the point near where the cylinder is applied towards the limits of the heart in every direction. So long as percussion is made over the body of the heart, a distinct sharp shock is felt directly in the ear; but as soon as the limits of the heart are passed, this sharp shock immediately ceases, even in passing from one solid organ to another in contact with it, as from the heart to the liver" (DRS. CAMMANN and CLARK). Practice will enable a discrimination to be made between the characteristic sound of the heart and the diffused shock produced by striking the ribs.

The mean diameter of the healthy adult heart (not the anatomical *præcordial* region only) has been found to be as follows:

		Inches.	Lines.
Vertical diameter,	Male,	4	0
	Female,	3	7
Transverse diameter,	Male,	4	4
	Female,	4	1
Right oblique diameter,	Male,	4	10
	Female,	4	10
Left oblique diameter,	Male,	3	10
	Female,	3	7

Generally the region of this *deep-seated dulness* extends transversely from the left nipple to a little to the right of the sternum, and vertically from the third to the sixth ribs. The true anatomical *præcordial space* may be diminished if the lungs are largely developed, and may disappear if the anterior margins of the lungs are emphysematous, so that their edges meet in front of the organ.

The region of the heart's superficial dulness is increased whenever the heart is enlarged, or whenever fluid to any amount is effused into the sac of the pericardium. If the walls of the ventricles are hypertrophied, or if their cavities are dilated, the dulness will extend over a wider surface, and its extent is in some degree a measure of the increased size which the organ has attained. The enlarged heart pushes aside the lungs, and a larger portion of it comes in contact with the parietes of the chest. In hypertrophy with dilatation the heart attains the largest size that it is capable of, and the præcordial region may yield a dull sound over a square surface of from two to six inches. When hypertrophy predominates over dilatation, the space which yields a dull sound is wider from above downwards. When dilatation predominates over hypertrophy, the region in which a dull sound is yielded transversely is wider. When there is fluid in the pericardium, a larger surface than natural in the *præcordial* region yields a dull sound; the degree of dulness is more pronounced, and the sensation of resistance considerably greater than in the former case.

The situation of the dulness, its amount and degree, are the guides as to its probable cause. When it is caused by enlargement of the heart, the site of the dulness is lower down and more to the left side than when it depends upon liquid effused into the pericardium. If a large amount of fluid is contained in this sac, a dull sound may be elicited by percussion as high as the articulation of the second rib with the sternum, and even in some cases as high as the first rib. The degree of dulness over fluid is also much more marked than over the heart itself, and the resistance to the finger is greater. Solidification of lung in the immediate vicinity of the heart will cause an apparent extension of the cardiac dulness.

Auscultation and the Sounds Associated with the Action of the Heart.—Auscultation of the heart, like percussion, may be either mediate

or immediate; but generally there are many objections to immediate auscultation. In the case of females it is indelicate; in dirty persons it is disagreeable; while in contagious diseases it is not without risk; besides, there are some situations in which either the ear cannot be applied, or in which the stethoscope is much more convenient. In cases of valvular disease of the heart the stethoscope, and especially the forms of the double stethoscope already described, are absolutely necessary in many cases.

The phenomena of the heart's action, as observed by the eye on the heart of a living animal exposed to view, or as appreciated by the ear and hand applied over the cardiac region of man, are of the most definite nature; and in determining the alterations of sound produced by disease, it is necessary to bear in mind the occurrence and sequence of the following incidents.

The two auricles of the heart contract at the same instant of time, and the contractions of the right and left ventricles are also simultaneous. The contraction of the ventricles follows immediately that of the auricles. The relaxation of the fibres of each part of the heart follows immediately their contraction, and a short but distinctly appreciable period of repose intervenes between the relaxation of the ventricles and the secondary contraction of the auricles. Each complete revolution of the heart is thus accompanied by two successive sounds, separated from each other by intervals of silence. These two sounds *are unlike;* and the two periods of silence *differ in duration.* Within the limits of health these sounds have a variable duration, and limit of surface over which they are heard, as well as of intensity, and of a certain *rhythm.* The *rhythm* of the heart is maintained when its two sounds succeed each other, and are followed by an interval of repose, which varies in length according to the duration of the previous systole, and according to the rapidity with which the sounds succeed each other.

The first sound is coincident with the systole of the ventricles, the impulse of the apex against the side of the chest, and the pulse of the large arteries. It is sometimes also called the *systolic* or *inferior* sound of the heart. It should be listened to over the apex of the heart.

The second sound is synchronous with the diastole of the ventricles, the recedence of the heart from the side, and the pulseless state of the large arteries. It is sometimes also called the *dias-*

tolic or *superior* sound. It should be listened to at *mid sternum*, near its left edge, and on the level of the second interspace.

A very short silence (which only becomes obvious when the pulse does not exceed sixty in a minute) succeeds the first sound; but a distinctly appreciable period of silence and repose, of some duration, succeeds the second sound.

The *first sound* is of a prolonged and dull character compared with the *second sound*, which is more quick, short, and clear, and bears a close resemblance to that produced by *lightly tapping, near the ear, the knuckle of a bent finger with the soft extremity of a finger of the other hand* (HOPE).

The Line of Transmission of these sounds respectively it is of the greatest practical importance to observe; because, being so constant in health, any variation indicates some modifying cause.

The *first sound* passes slantingly upwards to the left acromial angle, growing weaker and weaker on the way. Its intensity diminishes much more on the way to, and at, the right acromial angle. The propagation backwards of the first sound is thus clearest and fullest to the left—so that, while audible at the left back, it may be inaudible at the right.

The *second sound* has the region of the base for its centre; and in nine people out of ten is heard more clearly at mid sternum, *on the level of the second interspace*, than at any point of the præcordial region (WALSHE). It radiates towards the right and left acromial angles, and with greater clearness to the left than the right, while posteriorly it is heard at the surface with less clearness and distinctness on the right side than on the left.

Next to the lines of propagation of the sounds being determined as above indicated, it is necessary to analyze the sounds, and compare them—(1.) At both sides of the apex and region of the base; (2.) At base and apex on the same sides of the organ. (3.) At base and apex of opposite sides. In any one of these regions, compared with the other, the sounds in health are found to vary so materially in positive and relative properties that any single description of them cannot be given.

The natural sounds of the heart thus indicated may be of abnormal character, as regards *intensity, pitch, duration, quality, rhythm, reduplication*, and *apparent distance*, at each of the following points:—namely, at the third interspace, and along the third rib for the distance of three inches from the left edge of the

sternum, *for all sounds;* at the point where the apex beats, *for mitral sounds;* at the left of the sternum, and over the ensiform cartilage (if the apex be not in this position), *for tricuspid sounds;* at third left costal cartilage and adjoining part of the sternum, *for both aortic and pulmonary sounds;* at second right and second left costal cartilages, *for the sounds of aortic and pulmonary orifices respectively* (*What to Observe*, p. 43).

The term "*sound*," in reference to cardiac diagnosis, is understood to refer to the natural sounds of the heart, either normal or modified in character, as detailed above.

It is important to observe the relationship of the sounds of the heart to the pulse. The first sound anticipates, by a very short but appreciable interval, the pulse at the wrist. An interval consistent with health is undetermined; but it may be stated generally, that if the diastole of the most distant arteries, such as the posterior tibial, behind the inner ankle, or the arteries on the dorsum of the foot, is so much retarded as to become synchronous with the *second sound*, the state indicates disease. It is a frequent attendant on insufficiency of the aortic valves.

When the sounds of the heart, after a certain number of perfectly regular beats, are suspended during the time usually taken to perform an entire revolution of the cardiac functions, the sounds of the heart are said to *intermit.* A sudden pause or silence then occurs, and such an intermission sometimes happens more or less regularly, or after a fixed number of regular beats. This constitutes *intermittence* or *intermission* of the heart's action. It may occur in individuals otherwise in perfect health; but it is common in diseased states of the valves or orifices of the heart, where some impediment exists to the direct passage of the blood, or where regurgitation is permitted.

Morbid Sounds of the Heart, or Murmurs.—The term "*murmur*" is applied to a sound superadded to the normal sound of the heart, and which may occur with one or more of the natural sounds. The term was first employed by the late Sir John Forbes, and has been very generally adopted since. These may be so obscured, or even obliterated, that the *murmur* or *morbid sound* is alone heard. According to their supposed seat of production these *murmurs* are—(1.) *Endocardiac*, sometimes called *valvular;* (2.) *Pericardiac*, also called *exocardiac.*

All endocardiac or valvular murmurs yield a "*blowing*,"

"*rough*," "*rasping*," "*sawing*," "*booming*," or "*bellows*" *sound*, as in the whispered expressions of the words "*who*" or "*awe*," the double letter "*ss*," or the single letter "*r*;" and there are certain spots where they may be heard in their greatest *maximum force;* namely,—(1.) A few lines above the left apex; (2.) Just above the ensiform cartilage at mid sternum; (3.) On the level of the third interspace; and (4.) At the junction of the third left cartilage with the sternum.

The physical causes which may explain the *mechanism* of these murmurs are due either—(1.) To pure constrictions of natural orifices; (2.) To pure widenings of natural orifices; (3.) To pure roughness of surfaces; or (4.) To the association of the latter condition with either of the two former.

When murmurs are due to such single or combined mechanism, they are said to be *organic murmurs*, to distinguish them from *inorganic* murmurs due to certain morbid causes not yet well understood.

These *inorganic* murmurs are connected—(1.) With certain states of the blood, as in spanæmia; or (2.) With dynamic or functional action of the heart itself.

It is of the greatest importance to determine, therefore, the nature of a *murmur*, especially as to whether it is really *organic* or *functional.*

In this investigation the essential points to be inquired into, to guide the diagnosis, are as follows:—(1.) Observe the relationship of the murmur to the systole or diastole; that is, the *rhythm* of a murmur, or the position, in point of time, which it holds during the different physiological acts which constitute a complete cardiac pulsation—namely, the *contraction*, the *dilatation*, and the *period of rest* of each of the cavities; (2.) The spot of its maximum intensity on the surface of the chest; (3.) The direction in which the murmur is transmitted; (4.) Its quality and pitch; (5.) State of the natural sounds of the heart which may remain; (6.) Presence or absence of any audible phenomena in the arteries or veins, or both; and (lastly) The duration and clinical progress of the case.

Each orifice of the heart may be the seat of two murmurs, *constrictive* and *regurgitant, with* or *against* the current; and thus *eight murmurs* are the total number the occurrence of which is possible. The essential characters of these murmurs are con-

densed as follows from Dr. Walshe's *Practical Treatise on Diseases of the Lungs and Heart;* and from Professor W. T. Gairdner's paper, entitled "A Short Account of Cardiac Murmurs," *Edin. Med. Journal,* Nov., 1861.

In determining the attributes of a cardiac murmur, the first step in the inquiry is to determine which is the *second* sound of the heart, and which is the *first.* In the *rhythmical* succession of the heart's actions the phenomena which we can appreciate externally are a little later than the commencement of the heart's action. *Before there is either sound or impulse the contraction has already taken place;* and whatever the pathological origin or seat of the murmur may be, those which immediately succeed the first sound and the impulse correspond to the period of ventricular contraction; and those which succeed the second sound correspond to the period of ventricular dilatation.

Dr. Gairdner gives the following classification and account of them:—

1. An *auricular systolic* murmur is one which *precedes and runs up to the* FIRST SOUND of the heart, which is in all probability produced in one or other of the auriculo-ventricular orifices, inasmuch as it coincides with the forcible emptying of the auricles into the ventricles through these orifices. Its reasonable interpretation, therefore, is *obstruction to the current of the blood* ENTERING *a ventricle.* If the left auriculo-ventricular orifice is affected, the murmur will be found to have the character of a *mitral murmur,* and to have its area at A (fig. 14). If, on the contrary, the *tricuspid* orifice be obstructed, the murmur will occupy the triangular area C (fig. 14).

2. A ventricular systolic murmur *succeeds and runs off from the* FIRST SOUND; and it may be produced either in the *auriculo-ventricular* or in the arterial orifices. In either case it coincides with the *emptying* of the ventricles; and, therefore, if *auriculo-ventricular* in origin, it is *a murmur of regurgitation;* if, on the other hand, it is of *arterial origin,* it is a *murmur of obstruction.*

A ventricular systolic murmur may thus have four distinct solutions among organic valvular diseases. If the area be *mitral,* it is *a murmur of mitral regurgitation.* If the area be *aortic,* it is *a murmur of aortic obstruction.* If the area be *tricuspid,* it is *a murmur of tricuspid regurgitation.* If the area is that of the

origin of the *pulmonary artery*, it is *a murmur which indicates pulmonic obstruction.*

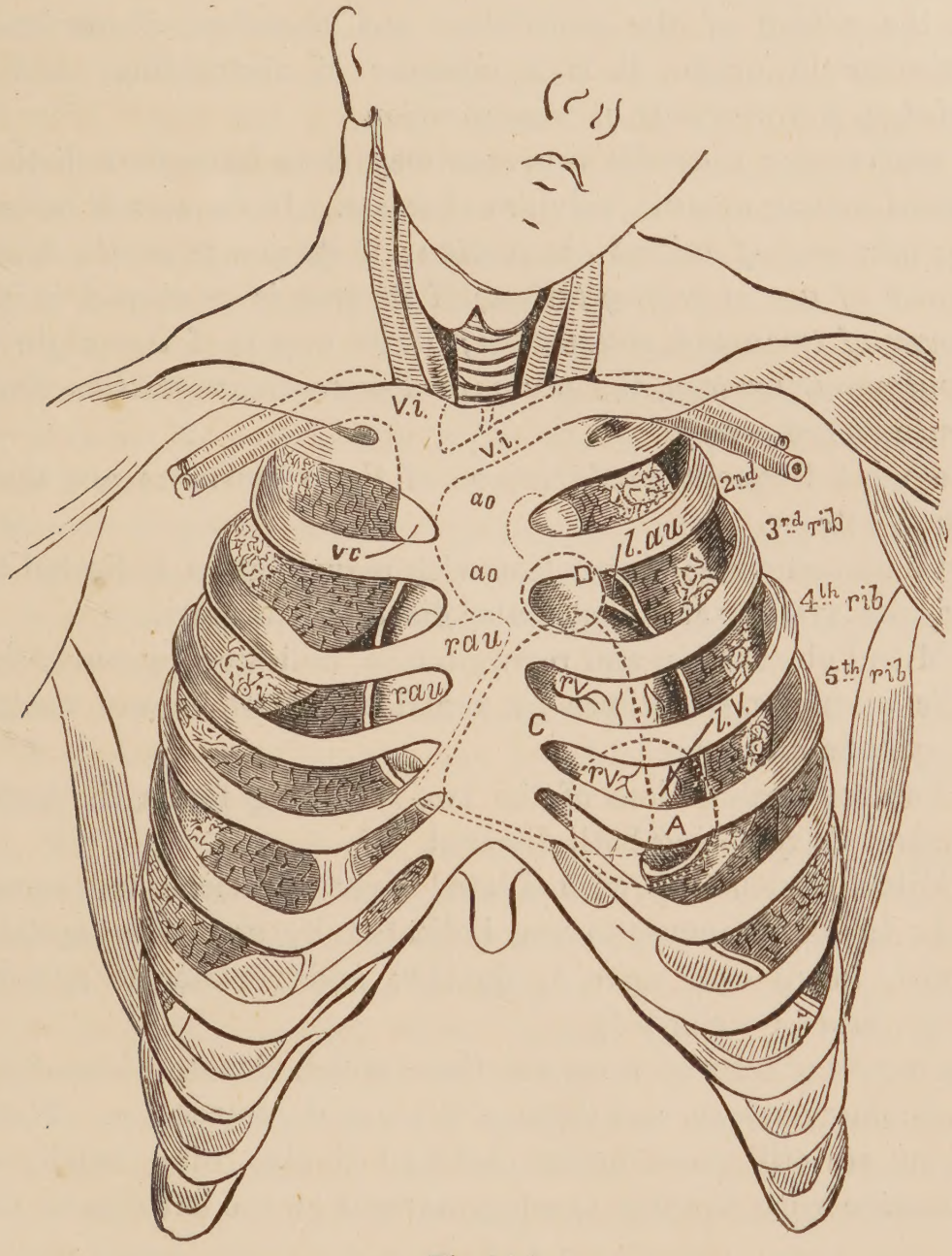

Fig. 14.*

* The Heart: its several parts and great vessels in relation to the front of the thorax. The lungs are collapsed to their normal amount, as after death, exposing the heart. The outlines of the several parts of the heart are indicated by very fine dotted lines. The area of propagation of valvular murmurs is marked out by more visible dotted lines. A, the circle of mitral murmur, corresponds to the left apex.

The broad and somewhat diffused area, roughly triangular, is the region of tricuspid murmurs, and corresponds generally with the right ventricle where it is least covered by lung. The letter C is in its centre.

The circumscribed circular area, D, over which the pulmonic arterial murmurs

3. A *ventricular diastolic murmur* succeeds and runs off from the second sound, and may be produced either in the *auriculo-ventricular*, or in the *arterial* orifices. In either case it coincides with the *filling* of the ventricles; and therefore, if *auriculo-ventricular* in origin, it is a murmur of obstruction; and if *arterial*, it is *a murmur of regurgitation.*

A *ventricular diastolic murmur* may thus have four distinct solutions among organic valvular diseases. If its area is *mitral*, it is *a murmur of mitral obstruction;* if its area is *aortic*, it is *a murmur of aortic regurgitation;* if its area is *tricuspid*, it is *a murmur of tricuspid obstruction;* if its area is of the origin of the *pulmonary artery*, the *murmur denotes regurgitation from the pulmonary artery.*

The most frequent combinations of these murmurs are those which denote—

1. Combined aortic obstruction with regurgitation, indicated by ventricular systolic and ventricular diastolic murmurs.

2. Mitral obstruction and regurgitation, indicated by *auricular systolic* murmurs, sometimes by ventricular diastolic and ventricular systolic murmurs.

3. Various combinations of the two preceding forms, the aortic and mitral valves being both diseased.

4. Mitral obstruction with dilated right ventricle, and consequently tricuspid regurgitation, indicated by *auricular systolic murmur*, heard over area A, fig. 14; and ventricular systolic murmur, heard over area C.

The rarest of all *murmurs* are those which denote *obstruction of pulmonary artery*, and those of *tricuspid obstruction.* These murmurs are still more rarely observed singly, being usually in combination with diseases causing murmur on the left side of the heart.

In a very able communication to the Hunterian Medical Society of Edinburgh, Dr. J. Warburton Begbie has recently called attention to "the diagnostic value of an accentuated

are commonly heard loudest. In many cases it is an inch, or even more, lower down, corresponding to the *conus arteriosus* of the right ventricle, where it touches the walls of the thorax.

The internal organs and parts of organs are indicated by letters as follows:—*r. au*, right auricle, traced in fine dotting; *ao*, arch of aorta, seen in the first intercostal space, and traced in fine dotting on the sternum; *vi*, the two innominate veins; *rv*, right ventricle; *lv*, left ventricle.

cardiac second sound" (*Edin. Med. Journal*, June, 1863). This accentuated second sound—equivalent to an intensified or greatly pronounced sound—is heard in instances of aortic aneurism and aortic dilatation, associated with atheromatous degeneration, as well as in some cases of hypertrophy and dilatation of the left ventricle. In a case of aortic aneurism the second sound of the heart has been observed so intensified or accentuated over the base of the heart as at once to be recognized even by *tyros* in the art of auscultation. When this sound occurs, which is of a *booming* or *ringing* character, it is to be presumed that the aortic valves are competent. If they were insufficient, a diastolic murmur would be the result, as the prominent physical sign, and apt to cause the most skilful physicians to overlook the existence of aneurism.

Modern diagnosis localizes murmurs chiefly from the observation of the areas of transmitted sounds already indicated. There are four distinctive areas to which murmurs arising at these orifices may be propagated.

The accompanying wood-cut (modified from those of Professors W. T. Gairdner, of Glasgow, and Luschka, of Tubingen), (fig. 14, p. 563), indicates the areas.

1. **Murmur connected with the Mitral Valve, Orifice, or neighbouring portion of the Left Ventricle,** may be the result of inefficiency of the valve, by changes in its structure, or from roughness of its edges, as by vegetations, shortening of the *chordæ tendineæ*, or fibrinous coagula amongst them. It may also result from simple roughness or deposit on the under surface of the valve without positive insufficiency. It is a *ventricular systolic murmur*, of maximum force, heard at and immediately above, or to the outside, *of the left apex*, and which may completely or partially cover the first sound of the heart at the left apex, but which may also preserve its natural characters towards the base.

This *systolic murmur* is faintly or wholly inaudible at the right apex, the mid sternal base, the pulmonary and aortic cartilages. It is more or less clearly audible about and within the inferior angle of the left scapula, and beside the dorsal vertebræ from the sixth to the ninth. This *murmur* is rarely of high pitch; and once established it is permanent.

To find the area of this murmur it is requisite to determine the exact seat of the apex beat, the patient lying a little to the left

side, or even on the face. If there is no distinct apex beat, find the most remote point downwards and leftwards at which the impulse of the heart is discernible; test this point by percussion, to observe if it corresponds with the margin of the cardiac dulness; test it also by auscultation, to hear if the *first sound* is conveyed thither with special distinctness. If a murmur concurs in position with the seat of these different phenomena, and if its seat of diffusion is *round this point nearly in a circle* (fig. 14), *it is probably of mitral origin.*

2. **Murmur associated with the Tricuspid Valve** may be due to regurgitation, or to the sharp collision of blood among thickened and roughened *chordæ tendiniæ.* It too is a *ventricular systolic murmur,* heard of maximum force *immediately above or at the ensiform* cartilage; inaudible, or nearly so, at the left apex, and very faintly, if at all perceptible, in the left vertebral groove opposite the lower angle of the scapula. It originates in the right ventricle; and when due to regurgitation, there is distension and pulsation of the auricle, vena cava, innominate and jugular veins, the distension of the latter being visible. It is generally a soft *murmur,* of low pitch, and rarely masks the systolic sound completely. It is a rare *murmur,* and often escapes detection from two causes—namely, a powerful mitral *murmur,* with which it is usually associated, or a deep-seated venous *hum.*

Professor W. T. Gairdner does not consider that *tricuspid murmurs* are rare, at least those of regurgitation. The area of tricuspid valve murmurs is over the right ventricle, where it is uncovered by lung—*i. e.,* at the lower part of the sternum, and over the whole space between this and the seat of the mitral murmur. It is usually but little audible above the level of the *third* rib, and is thus distinguished both from the pulmonic, and still more from the aortic murmur. Its area is indicated by the triangular space in fig. 14; but in cases of considerable hypertrophy and dilatation of the right side of the heart, especially in connection with emphysema (when the ventricle pulsates in the epigastrium), the murmur is heard loudest towards the xyphoid cartilage, and along the margin of the seventh left costal cartilage.

3. **Murmur connected with the Aortic Valve** habitually signifies a rough constriction of that orifice, and in rare cases has been

traced to fibrinous coagula impeding the egress of the blood. It likewise is a *ventricular systolic murmur*, heard of maximum force at *mid sternum, opposite the third interspace, or upper part of the fourth rib.* It abruptly loses force between this point and the left apex, where it may be almost inaudible. Faintly perceptible at the second left cartilage, it is clearly audible at the second right cartilage, the notch of the sternum, and the left vertebral groove, opposite the second, third, and fourth dorsal vertebræ, thence rapidly losing strength downwards. It originates at the aortic orifice, and disappears about the sixth dorsal vertebra. It is propagated into the arteries of the neck. It is a high-pitched, harsh, loud, and prolonged *murmur.* The concurrence of ventricular hypertrophy increases its intensity and prolongs its duration the more contracted the orifice is. The area of this murmur corresponds generally to the regions of the sternum, and is often absolutely loudest close to the xyphoid cartilage.

4. **Murmur connected with the Orifice of the Pulmonary Artery** may indicate obstruction or simple roughness in its valves, or pressure on the vessel by adventitious masses in the pericardium. It is a *ventricular systolic murmur*, heard of maximum force at the *sternal edge of the third left cartilage,* or a little lower down, and imperceptible in the back. It is rarely met with.

5. **The Murmur indicative of Obstructive Narrowing of the Mitral Valve** is a *ventricular diastolic murmur*, heard in maximum force *immediately above and about the left apex.*

6. **The Murmur which indicates the probability of Tricuspid Narrowing or Obstruction** is also a *ventricular diastolic murmur*, and is heard in maximum force at the *ensiform cartilage.*

7. **The Murmur which indicates Regurgitation at the Aortic Orifice** is likewise *ventricular diastolic,* and is heard of maximum force at mid sternum, opposite the third interspace or fourth cartilage; and it is often carried down loudly to the left apex. It is usually of an inspired blowing character, sometimes almost hissing, rarely rough, and completely fills up the interval of repose and silence which ought to follow the second sound. It differs from *constrictive* aortic murmur in being heard with almost as much intensity *about the ensiform cartilage as opposite the third interspace.* When it covers completely the second sound of the heart at the point of its maximum intensity, the valves may be presumed to be utterly incompetent.

8. **The Diastolic Murmur connected with insufficient Pulmonary Valves** is so rare that it is only mentioned here to complete the notice of cardiac murmurs which may be heard.

The relative frequency of intra-cardiac organic murmurs Dr. Walshe states to be in the following order, commencing with the most common,—namely, *mitral regurgitant; aortic constrictive; aortic regurgitant; mitral constrictive; tricuspid regurgitant; pulmonary constrictive; pulmonary regurgitant; tricuspid constrictive.* These may, however, be variously associated together; and when they co-exist, they are to be distinguished by the *rhythm*, the *pitch*, and the *character* of the aspiration

The point at which a murmur is produced being in the majority of cases one of the four valvular orifices, all doubtful murmurs should be tested in the first instance on the supposition that they are valvular. With this view the most important practical points to be determined are,—(1.) The actual size and position of the heart, and the relation of its several parts to the thoracic walls (as described in the previous sections); (2.) The anatomical *præcordial* space must especially be accurately defined; (3.) The exact point of the apex beat is to be determined; (4.) The character of the impulse both of the right and left ventricle should be carefully studied; (5.) Determine by careful stethoscopic observation the exact seat and the limits of the diffusion of the murmur actually under observation.

The Pericardial Murmurs consist of *friction* or rubbing sounds, analogous to those already described in the pleuræ (Table, p. 543), and result from the movements of two opposed surfaces on each other, having been rendered dry or rough by change of tissue or exudation. Pericardial murmurs are almost limited to cases of inflammation of the pericardium. These friction murmurs are generally double, and are sometimes louder during the diastole than the systole of the ventricles. They appear to be superficial or near, and are seldom audible beyond the limits of the *præcordial* region. They never replace the ordinary sounds of the heart, and are entirely independent of them. Their duration is usually short, frequently ceasing entirely after having been heard for a few days, and not unfrequently changing their character and seat within the period that they are audible. A peculiar vibratory thrill, sensible to the hand laid upon the parietes, frequently accompanies them.

Section VIII.—Significance of the Pulse in Cardiac Disease.

Certain forms of cardiac disease are capable of impressing peculiar and well-marked characters upon the pulse. The pulse ought to correspond with the ventricular systole, and with the first sound of the heart; and when the heart, the arterial system, and the blood are each in a normal condition, the force, the strength, the frequency, and the fulness of the radial pulse may be taken as a measure of the strength or feebleness of the systole of the left ventricle, of the rapidity with which the movements of the heart are performed, and of the amount of blood transmitted at each systole of the left ventricle. The beat of the pulse in the radial artery ought to be a little—a very little—later than the ventricular systole. The interval is almost imperceptible, unless the pulse is unusually slow. In the dorsum of the foot the interval is more easily appreciated. It is advisable to place one hand upon the præcordial region, or to auscultate the region of the heart, while the finger is on the radial pulse, to determine these points in all cases.

In softening of the heart the pulse is sometimes much less frequent than the cardiac systole, because the impulse fails to be transmitted (Fuller, J. C. B. Williams, Gairdner).

In hypertrophy of the left ventricle, when the parietes are increased in thickness, the systole is strong in proportion, the blood is propelled into the aorta with increased force, and the radial pulse is strong and hard. Its velocity may not be increased, but the systole takes a longer time to be completed, and the pulse will "dwell longer under the finger." When dilatation is combined with hypertrophy, so long as the circulation continues free, the pulse will be full, or of larger volume, because the amount of blood propelled at each systole will be larger; but when dilatation is combined with attenuation, or if dilatation simply prevail, the radial pulse will have nearly opposite characters to those stated. It will be soft and weak.

The pulse in aortic regurgitation acquires a peculiar character. It is jerking and receding, though regular; while the pulsations of the arteries of the upper extremities and of the neck are visible, as if "leaping." It has been named a "locomotive" pulse

by Bellingham and Todd—*i. e.*, the arterial tubes are SEEN to move by elongation—"leaping forth at each beat of the heart." This is sometimes termed the "pulse of unfilled arteries." In well-marked examples it appears as if the blood was divided into separate little balls, which pass in rapid succession under the finger. The sensation is better *seen* and felt in a large artery, such as the brachial, and two or more fingers should be laid on the line of artery. The arteries, when tortuous especially, appear like worms under the skin, wriggling into tortuous lines at each pulse (WILLIAMS). This kind of pulse is also sometimes observed in *aneurism of the ascending or transverse portion of the arch of the aorta*, as well as in cases of disease of the aorta itself, when it has become rigid, elastic, and dilated.

Intermission of the pulse indicates the slightest degree of derangement of the heart's action. It is not uncommon in persons advanced in life, in gouty subjects, and in derangement of the digestive organs with flatulence. It is also met with in cases of disease of the valves, or of the muscular tissue of the heart.

An unequal pulse is one in which some of the pulsations are strong, and others weak.

An irregular pulse is one in which a few rapid beats are succeeded by one or more slower beats, and when the interval between them is different.

An unequal and irregular pulse are much more unfavourable signs than a simply *intermittent pulse.* Both are met with in the same cases—in certain diseased states of the valves at the left side of the heart, or in morbid conditions of the muscular tissue.

In contraction of the left auriculo-ventricular orifice, the pulse, in addition to being weak and intermittent, will become small, irregular, and unequal, although the heart's action continues to be strong. "The heart may often beat so violently as to shake the patient in his bed, while the pulse is small, weak, and irregular. It appears as if there were two *pulses;* one is slow and deliberate for two or three beats, succeeded by three or four rapid and indistinct pulsations" (ADAMS).

In mitral regurgitation, when the closure of the valve is very imperfect, the pulse becomes weak and small, and will intermit if the circulation is hurried; and when a considerable quantity of blood is permitted to regurgitate into the auricle, the pulse will also become irregular and unequal.

In contraction of the aortic orifice (when it becomes extreme only) the pulse becomes small, and intermittent or irregular, resembling the pulse of considerable constriction of the mitral orifice. When the contraction is slight, it is neither weaker nor smaller than natural, and is perfectly regular.

In degeneration of the muscular tissue the pulse in the advanced stages is small, weak, irregular, and unequal, sometimes slow, and the impulse of the systoles fails to be propelled.

In pericarditis with copious liquid effusion, the pulse presents somewhat similar characters.

During the formation of fibrinous concretions within the cavities of the heart the pulse suddenly becomes small, weak, intermittent, and irregular.

Section IX.—Use of the Sphygmograph.

The *Sphygmograph* was originally devised by M. Marey to determine various points in the physiology of the circulation of the blood; and, as an instrument of the greatest accuracy, it could not fail to become of great value as an aid in determining the nature of diseases of the heart and arteries. The instrument and its practical use was first shown me in the autumn of 1863, by my teacher, the late John Goodsir, the distinguished Professor of Anatomy in Edinburgh, who predicted for it an extensive field of usefulness in diagnosis. Dr. Anstie (who was the first to show the instrument to a medical society in this country), and Dr. B. Foster (who was the first to publish an account of any considerable number of observations made with the instrument), and Dr. B. Sanderson, have since been the most active exponents of the methods of appreciating the characteristics of the pulse by the sphygmograph. From the writings of these men, but more especially from those of Dr. Foster, of Birmingham (who kindly lent me his MS. notes), the following account of the sphygmograph is given.

In the conventional routine of "*feeling the pulse*," the unaided sense of touch is quite unable to appreciate many characteristics which the sphygmograph enables us now to appreciate; and a study of its practical application cannot be dispensed with. In place of its being able to take the place of "*feeling the pulse*," it will not only extend our practical know-

ledge derived from this ancient method of observation, but it will define and greatly extend our knowledge of the more delicate characteristics of the pulse which are beyond our sense of touch to appreciate. To "*feel the pulse,*" and to "*look at the tongue,*" all

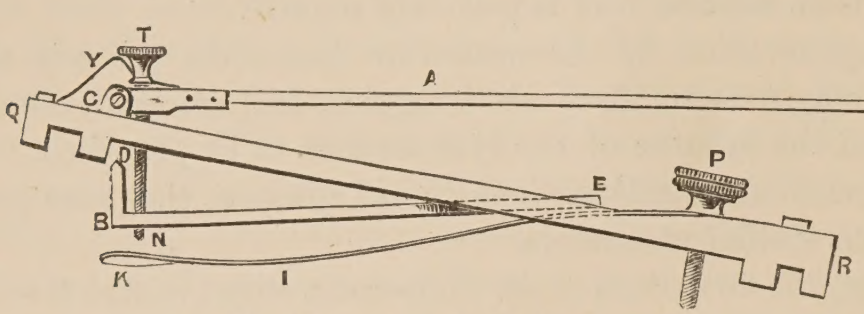

Fig. 15.*—Marey's Sphygmograph.

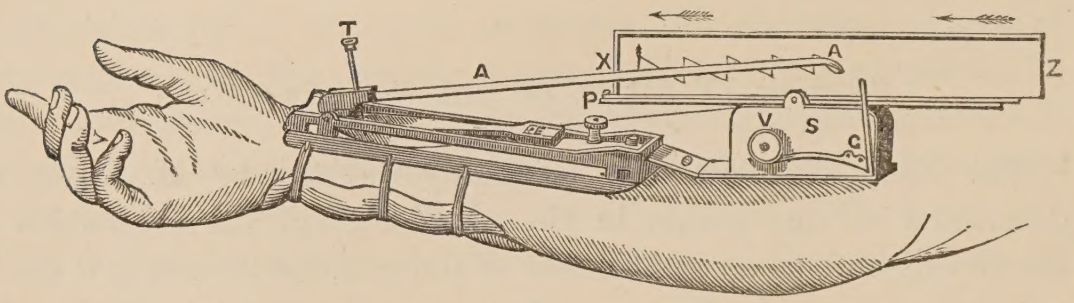

Fig. 16.*—Sphygmograph applied to the arm.

patients consider as most essential duties on the part of the physician; and the sphygmograph does not aim at superseding either of these essentials. It aims at adding to our knowledge by writing down, as it were, for our inspection the traces of

* The wood-cut shows in the interior of the frame Q R the essential part of the instrument, which consists of a flexible steel spring, I, covered on its under surface at its free extremity with a convex plate of ivory, K. This ivory plate rests upon the artery to be examined, and, by virtue of the elasticity of the spring I, exerts a certain pressure upon it. Each pulsation of the vessel raises the spring slightly at K, and the multiplication of this movement is obtained by means of a very light lever, A, which moves upon a pivot, C. The elevation of the spring is transmitted to the lever, very near to its centre of movement, by means of a bar of metal, B E, which moves round the point E; this bar terminates in a vertical plate, B D, and is pierced by a screw, T. When the screw acts upon the spring, the connection is established between the spring and the bar, and the movements of the spring are transmitted to the bar, and through its vertical plate to the lever. In order to insure the transmission of the movement, the plate B N must be in contact with the under surface of the lever; by means of the screw T we can arrange this, and regulate the interval between the point of the plate B D and the under surface of the lever. In order that the lever should not be projected too much upwards by sudden movement, and also that it should overcome any slight friction experienced in the paper at its terminal point A, a small spring, Y, rests upon its

hardness or softness of the pulse, as a measure of *arterial tension* or of *feeble pressure of the blood within the blood-vessels.* The value of the instrument at present mainly rests on the exactitude and precision which it gives to our notions regarding "the *practical significance of various forms of pulse*"—a kind of knowledge only acquired by great and long practical experience, and a kind of knowledge very difficult, if not impossible, to convey by a teacher to a pupil.

The sphygmograph, when in action, gives an exact representation of the pulse-form—the frequency of the pulsations, and their regularity. It enables us to see at a glance any peculiarity in the entire series, or in any single pulsation. A trace, as below, is

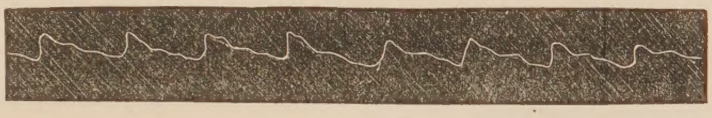

Fig. 17.

composed of a series of curves, each of which corresponds to a complete revolution of the heart, and is called a *pulsation.* Each pulsation is composed of three parts—the line of *ascent*, the *summit*, and the line of *descent.* The line of *ascent* is produced during the flow of blood into the arterial system after each cardiac systole; and tells us, by its form, the manner in which the blood enters the vessels. The more rapid the afflux, the more quickly the pressure in the arteries will be elevated, and the more vertical will be the ascent of the lever. When the entry of the blood is slow, the line of ascent will be traced by the lever obliquely, sometimes in a curved form. The line of ascent,

fixed extremity, and presides over its descent. The screw, P, enables us to regulate the amount of pressure exercised upon the artery by the spring, I.

The wood-cut (fig. 16), modified from Marey, shows the instrument placed upon the arm over the radial artery in the position for use. The lever, A, is here seen to carry at its free extremity a little pen, which, filled with ink, registers its movements upon the paper which covers the plate X Z; this plate is moved at a uniform rate in the direction indicated by the arrows by means of watch-work placed beneath in the case, S. Ten seconds are occupied by the passage of the plate. The button, V, enables us to wind up the watch-work; and the small regulator, G, starts the plate, or stops its motion, as desired. The application of the instrument Dr. Foster has found much facilitated by the use of elastic bands, instead of a silk lace, as recommended by Marey. These bands embrace the arm, and are hooked on to the small projecting points on the metal framework, as seen in the diagram. The addition of a pad, suggested by Mr. Waters, to the under surface of the arm, renders the instrument more easy to the patient, and prevents any pressure from the bands.

in certain morbid conditions, exhibits a mixed form—the first part of the trace being vertical, the latter part curved. The *summit* of the pulsation corresponds to the duration of the arrival of blood in the artery, and designates the period during which the entrance balances the onward flow—in other words, the afflux and efflux are exactly equal. This period varies in length: it may be so short, that the summit becomes a mere mathematical point between the lines of ascent and descent; or it may be so long as to render the summit a horizontal line of some length. In the latter case, the lever traces the horizontal line, whilst the entry of blood into the vessel and its passage onwards mutually balance one another, and this line indicates the duration of the cardiac systole. The summit of the pulsation, when of any length, is not always horizontal; it may be formed by an ascending or descending plane, according as the afflux predominates over the onward flow, and *vice versâ*. The line of *descent* corresponds to the fall of the pressure in the arterial system, and is synchronous with the interval between the closure of the sigmoid valves and the next ventricular contraction. This line, by its obliquity, marks the celerity of the fall of the pressure within the vessels, and indicates the facility with which the blood passes on in its course. The amplitude of the pulse is indicated by

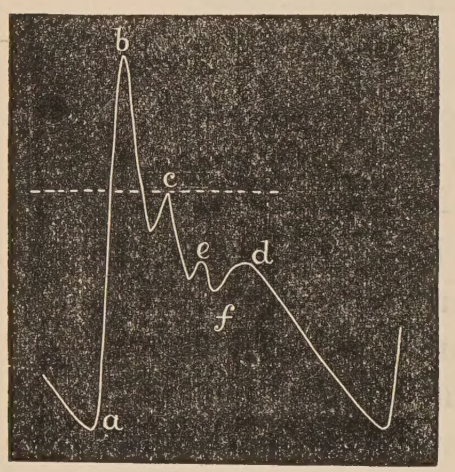

Fig. 18.*

* A typical radial pulse-trace, enlarged (after Dr. Foster). (*a* to *b*.) Line of ascent; (*c*.) First secondary wave; (*d*.) Second secondary wave, or true dicrotism; (*e*.) Third secondary wave, not generally seen. The notch in which *e* is placed corresponds to closure of the aortic valves. The first secondary wave, *c*, should be situated at the junction of the *upper third* with the *lower two-thirds* of the line of ascent, *a*, *b*, about the level of the dotted line. From *a* to *f* the ventricle is contracting or contracted, and *f* marks the closure of the aortic valves. The fourth wave, *e*, is seldom marked, but the three others can generally be distinguished, except when the tension is very high. The line of ascent corresponds to the ventricular systole, and marks the flow of blood into the aorta and great vessels.

The line of descent is generally broken by the occurrence of several secondary waves, which correspond to the vibrations in the blood column, alternately to and from the heart. The summit of the trace is usually followed by a small notch, and then the trace rises again to form the first secondary wave, *c*. The relative position of this wave to the summit part is of most practical importance to observe.

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the obliquity of this line. The form may vary very much; sometimes it is purely oblique, at others a curve convex upwards, and occasionally one or more undulations may be seen in it. This last peculiarity is often very marked, and in some cases is perceptible to the finger; it has been termed *dicrotism.* By this term we should only understand undulations occurring *in the line of descent.* Similar peculiarities have been pointed out in organic diseases of the heart; but in these cases the dicrotism belongs to *the period of ascent,* and is due to very different causes. The sphygmograph enables us to recognize in which period the peculiarity occurs. Thus, conditions which are associated with great elasticity of the arterial walls, as dilatation of the vessels and easy passage of blood through the capillaries, are marked by evident dicrotism in the line of descent. The following trace (fig. 19), taken during the perspiration of hectic shows this characteristic.

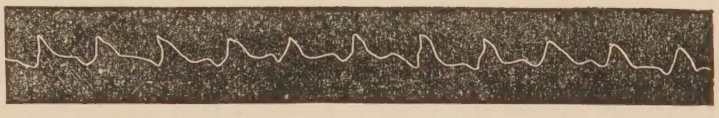

Fig. 19.

Senile change (fig. 20) in the vessels, and consequent loss of elasticity, is indicated by,—(1.) The absence of dicrotism; (2.) By the great dimensions of the curve; (3.) By the closer proximity, not only of the first but of all secondary ascensions to the apex of the curve; (4.) By the extreme predominance of the first secondary wave; and in near relation to this pulse of old age stands the radial pulse of people not far advanced in life, and suffering from hypertrophy of the left ventricle; and next, that which accompanies insufficiency of the aortic valves.

The deep notch in which the *third* secondary wave, *e*, is situated, and which precedes the *second* secondary wave, *d*, is of great importance, as it marks the closure of the aortic valves. It corresponds with that reflux of blood towards the heart which forces together the lappets of the valves; and the *second secondary* wave (or dicrotism proper) corresponds to the vibrating motion towards the periphery given to the blood by the sudden flapping together of the aortic valves, and the prevention of regurgitation into the left ventricle.

From *a* to the deep notch preceding *d*, corresponds to the period of the heart's systole, and measures the duration of each cardiac contraction. From the deep notch preceding *d* to the beginning of the next line of ascent marks the period of the heart's diastole (Dr. Foster's *MS. Notes*).

The great parts to be noticed in the senile pulse are the high positions of all the secondary waves in the line of descent, and the enormous size of the first secondary wave as compared with the second secondary wave or true *dicrotism*.

Fig. 20.

In examining a pulse-trace, one should note, in addition to the form of each pulsation, whether the summits of all of them can be joined by a straight line, and whether the bases can be also connected by a similar line parallel to the former. In some instances this ceases to be the case, and a series of pulsations cannot be contained between such imaginary lines. The pulsations become irregular, and the line to join their summits or bases must cease to be horizontal. The line joining the summits of a series of pulsations is the line of the maxima of *arterial tension*. Its value as an indication is not absolute; it only tells us the variations that the arterial tension may undergo during the period of the observation; and it enables us to judge of the relative pressure within the vessels during any of the cardiac contractions registered. This line of greatest tension is of much value, and, with the corresponding line of least tension, should be observed in all cases; as these lines generally undergo parallel deviations, and a glance at either usually suffices to inform us of any change. Different *diseases* cannot be recognized by pulses peculiar to them; but the pathological changes which the normal curve of the radial artery undergoes take place simultaneously with the changes indicated by the rise or fall of temperature in fever (Wolff).

The *frequency* of the pulse may be studied by means of the sphygmograph; for, as the plate moves at a uniform rate, and occupies exactly ten seconds in its passage, we can with ease calculate the pulse-rate. Slight variations in frequency, and irregularities that would most probably escape appreciation by the unaided touch, are by this means revealed. The frequency of the heart's action, according to the French physiologist, depends very much upon the state of the circulation in the vessels of the periphery—an easy passage of blood favouring the increased action—a difficult passage (by reason of the greater arterial

tension) causing diminished frequency of the ventricular systole. The law is thus laid down by Marey that, in the majority of cases, "*the frequency of the pulse*—i. e., *the number of contractions of the heart—varies inversely to the arterial tension;*" but Onimus and Viry maintain that "*the number of contractions of the heart varies directly with the initial force.*"

Even if a pulse-trace fails to indicate any specific lesion, the sphygmograph is the most exact measurer of tension and index of a *hard* or *soft* pulse. When we find the pulse-trace without any well-marked notch *before* the second secondary wave, and the line of descent forming an almost unbroken oblique line, it indicates *high arterial tension,* with arteries unusually full, giving the free, incompressible hard pulse. On the other hand, when the line of descent falls suddenly, the arteries are insufficiently filled, and the pulse is known as the soft compressible pulse.

The *force* of the pulse is indicated by the height of the pulsations. The greater the elevation of the lever, the greater the energy of the pulse-beat; and we may say that, in many cases, the strength of the ventricular contraction is expressed by the force of the pulse. This law, however, has many exceptions; and we find that the altitude of the pulse-trace depends on several other conditions. For example :—

(1.) The volume of the artery greatly influences the amplitude of the trace. This can be well seen in traces collected from old persons. In senile changes (fig. 20) the volume of the vessels is increased considerably, and the trace betrays great fulness. Marey believes this to be due, not solely to the hypertrophy of the ventricle which exists in the old, but also to the dilatation of the artery (fig. 20).

(2.) The state of arterial tension modifies greatly the force of the pulse; and, as the tension is dependent on the state of the capillary circulation, it may be said that in most cases "the force of the pulse is not in relation with the energy of the ventricular systole, but that it is regulated by the state of the circulation in the ultimate ramifications of the vascular system." By means of the manometer, in a great number of experiments this law has been proved to hold good—a feeble state of arterial tension giving to the finger and the instrument the sensation of increased amplitude. The following diagrams (figs. 21 and 22) illustrate this (MAREY).

The difference in the amplitude of the traces is very distinct. In the state of feeble tension, or easy passage of the blood

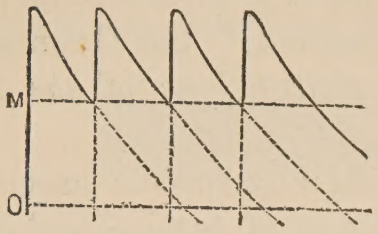

Fig. 21.*

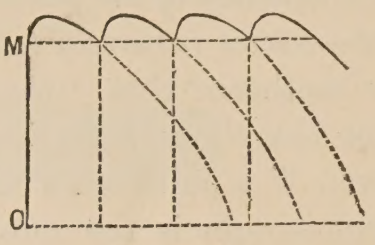

Fig. 22.†

onwards, the lever falls quickly to the point of least tension, and is elevated considerably at each pulsation. In the case of difficult passage of the blood through the capillaries, and consequently of great arterial tension, the lever descends slowly by a line convex upwards; and, long before it has reached a minimum tension equal to that in the former case, the lever is raised slightly by the next pulsation. While the lines of the maxima of arterial tension are the same in both cases, the lines of the minima are very different. On this depends the amplitude of the pulse-trace.

(3.) The duration of the interval which separates the pulsations has also a distinct amplitude of the trace. This is due to the fact that, during a long interval, the blood flowing continually onward lessens the pressure in the vessels, and thus favours the greater amplitude of the next pulsation. This is well seen in a trace (fig. 23) taken from a patient of Dr. Foster's in the Queen's Hospital, Birmingham.

Fig. 23.

The condition of the vessel itself, as to permeability below the point observed, influences the force of the pulsation by altering the pressure within the artery. Marey has also pointed out that in some cases, where the pulse-beat is almost imperceptible to the finger, the sphygmograph records a considerable amplitude of trace; and *vice versâ*. Such cases have been associated with a very slow distension of the vessels.

* The form of pulsation in a state of feeble tension.

† Under a state of strong tension.

But it is now shown that the modes of adjusting the instrument have a great influence on the results. For example, *by a more accurate apportionment of the pressure of the tactile spring to the power of the pulse,* very much larger and more developed curves can be obtained. Under certain circumstances, also, Dr. Anstie has shown that alterations of the weight of the writing lever may greatly increase the size of the curves obtained—a heavy lever suiting a strong pulse, and a light lever a weak one. Drs. Beigel, Sanderson, and Anstie have each shown the advantage of suspending weights in such a position on the writing lever as to exercise more or less pressure; or of weakening the spring by placing pledgets of lint or pieces of wood under the brass work, so as to ease off the pressure in taking the traces of very weak pulses. Also, Dr. Anstie points out that the highly significant trace of delayed systole (represented by the square-headed trace) may be spuriously manufactured by the instrument under either of two circumstances—namely, (*a.*) Too great friction between the pen and paper; and (*b.*) Too great resistance by the check-spring. This check-spring Dr. Anstie considers quite useless.

The discrimination of increased arterial resistance, as measured by the exaggeration of the systolic expansion, is the element of diseased action, whose detection and estimation, according to Dr. Sanderson, are the most important purposes to which the sphygmograph can be put. "Anatomical researches lead us to believe it probable that the earliest beginnings of what we may call degenerative disease consist in such structural alterations of the minutest arteries as, by rendering them less pervious to the circulating blood, must inevitably lead to increased arterial resistance estimable by the sphygmograph; and if by such an examination," continues Dr. Sanderson, "we can ascertain that the heart is over-taxed long before any change can be detected by auscultation or percussion, it is obvious that we have made a step forward in practical utility." Thus, he considers it likely that the *sphygmograph* is to be of greater use as an aid in forming an opinion as to the probable duration of life than in any other department of medical practice. There are many persons in whom, in the absence of any other trace of ailment, the pulse-curve indicates that the arterial resistance is excessive. The question is, "Are such persons sound?"

Dr. Anstie, on the other hand, is of opinion that the diseases

in which the *sphygmograph* will prove of the greatest diagnostic value are—(*a.*) In aortic regurgitation, by estimating the amount of valvular imperfection; (*b.*) In discovering unsuspected commencing cardiac hypertrophy, senile disease of arteries, or capillary disease, dependent on degenerative processes in the ultimate tissues; (*c.*) Above all, in discovering the existence of intra-thoracic aneurisms, and in deciding the locality of an aneurism; (*d.*) In aiding prognosis and decisions as to treatment in the course of acute diseases. Dr. Anstie has already made a series of observations in fever, pericarditis, pneumonia, and delirium tremens, which show that in this direction the sphygmograph promises to be of great value, as the best indication of the use or otherwise of certain remedial agents.

Section X.—General Symptoms of Thoracic Disease.

The elucidation of some of the topics, of which a short outline has been given in the previous sections, claims for the name of Laennec an immortal fame. He discovered how, *by means of auscultation,* disease might be detected. He not only accurately described the sounds heard in diseased states, and compared them with the sounds in health; but by morbid anatomy, in connection with careful clinical observation, he traced the mechanism of those sounds to anatomical and physical conditions of the organs with whose functions they were connected. The immediate effect of this inestimable discovery was to divert the attention of the physician from the study and observation of those vital symptoms and general states of the constitution so pregnant with information when correctly observed and properly appreciated. Now, it was the close observation of these vital symptoms, watching the order and the periods of their manifestations, and the modes in which they were combined, that distinguished the successful practice of our forefathers in medicine—such men as Cullen and the two Gregorys of former days. Disastrous results of treatment were the consequence of the circumscribed study of so-called physical diagnosis; "but what was lost in lives was gained in pathology." The physician of the present day now knows better. He is a close observer of vital symptoms, of constitutional states, and of physical signs; and with all the delicate instrumental

appliances to appreciate those signs, he forms his judgment from the combined evidence of them all.

The general symptoms which express derangement of the pulmonary organs and their functions are *dyspnœa, cough, expectoration, tenderness,* and *pain.* The general symptoms which indicate derangement of the cardiac apparatus are *palpitations, sinking,* and *fainting,* combined sometimes also with *dyspnœa, cough, pain,* and *tenderness.*

The sensation of *dyspnœa* is brought about by an embarrassed or laborious breathing, amounting in severe cases to a sense of suffocation, expressed by the common English phrase, "*want of breath;*" or by the meaning conveyed by the Latin term "*anxietas,*" when the dyspnœa is at the point of greatest intensity. It is aggravated by exertion, some positions of the body, and a full stomach. The act of speaking is frequently arrested "*to fetch a breath;*" and the patient who suffers from dyspnœa cannot hold the breath, or refrain from the attempt to inspire, as a person in health can. The dyspnœa may occur in paroxysms, and the acts of respiration may be painful. The difference between the dyspnœa of asthma and that of cardiac disease has been already given. (See p. 159, *ante,* as it is of extreme importance.)

The number of respirations performed in a given time is greatly increased, and often unequally so, when the paroxysm is aggravated. In health, from eighteen to twenty acts of respiration are unconsciously completed in a minute, according as a person is lying, sitting, standing, or walking; and the ratio of the acts of respiration to the pulse varies in the proportion of one to four (WATSON), or one to nearly six (WILKS); that is, about one complete act of respiration for every *four* or *six* beats of the heart. But so intimately are the functions of the heart and lungs dependent upon each other, that any deviation from these proportions, in the acts of the one or other set of organs, immediately influences the actions of the other.

Cough, expectoration, and the nature of the *sputa,* furnish valuable indications of thoracic disease.

Microscopical Elements of Sputa consist of—(1.) Young epithelium-cells—*i. e.,* of mucous corpuscles; (2.) Of mature epithelium in the form of pavement, cylindrical or ciliated bodies; (3.) Cells containing granules, or bodies like cells made up of granules; (4.) Pus-cells; (5.) Coloured corpuscles of the blood; (6.) Fibrin,

either in the form of flake-like membranes or in the form of casts of the smaller bronchi and pulmonary air-cells, as in the expectoration of pneumonia. They are sometimes seen as dichotomous cylinders with rounded enlargements, composed of fine filaments, generally covered with granules. They may be met with from the third to the seventh day in pneumonia. (7.) Fat occurs in granules or globules; (8.) Tubercle matter, earthy, calcareous, amorphous, and crystalline particles also occur; (9.) Substances derived from the food; (10.) Carbon and true pigment, free or contained in cells; (11.) Fragments of pulmonary tissue.

Chemical Characters of Sputa.—Much attention has not yet been given to the chemical characters of the sputa. Professor Laycock, of Edinburgh, had a chemical analysis made of very fetid expectoration in bronchitis, which demonstrated the existence in it of *butyric* and *acetic* acids. The odour was characteristic of the *butyrates of ethyl*, resembling the smell of may-flower or apple blossoms, combined with an odour of fæces. Chemical investigation may thus demonstrate the cause of the excessive fetor in those cases which resemble gangrene of the lungs, if the smell alone is considered (*Med. Times and Gazette*, May, 1857, p. 480). A much more extended analysis has been recently made by Bamberger into the chemistry of the sputa—(1.) In chronic bronchial catarrh; (2.) In bronchial dilatation; (3.) In chronic pulmonary tuberculosis; (4.) In the infiltration of acute tuberculosis; and (5.) In pneumonia. In sputa which is chiefly catarrhal the salts vary but little—the organic matters vary considerably. The insoluble salts form about 4 to 5·5 *per cent.* of the whole saline contents; the chief amount consists of *chloride of sodium* and *phosphate of potash.* Puriform matter predominating, causes the sputa to contain a greater quantity of organic and inorganic substances; there is considerably more *phosphoric acid* in the ash, considerably less *chlorine*, and less *sulphuric acid.* The ash of pneumonia sputa differs from that of catarrhal in several respects. The alkalies, combined with phosphoric acid (which amount to 10 and 14 *per cent.* of the saline constituents of catarrhal sputa), are almost entirely absent in pneumonia during the inflammatory period, but the sulphuric acid is remarkably increased. The quantity of chlorine (37 *per cent.*) is nearly the same as the average in the catarrhal (36 *per cent.*); and there is not much variation in the insoluble salts, except in the phosphate of iron

derived from the blood. In the period of resolution the sputa of pneumonia become more similar to the catarrhal; the *phosphoric acid* increases, the *sulphuric* diminishes, and the chlorine reaches a very high amount, while the potash and soda are present in the same relative proportion as in the catarrhal, whereas during the inflammatory period this was inverted. Sugar has been detected during the height of the inflammation; and Dr. Beale has shown that an excess of the chloride of sodium is constantly present in pneumonic sputa. In the sputa of bronchiëktasis, sulphureted hydrogen, acetic, butyric, and probably formic acids were detected (*New Syden. Society Year-Book*, 1860, p. 128; Schmidt's *Jahrb*, band 114, p. 3).

Sputa Typical of Pneumonia is characterized by its viscidity, semi-transparency, and tenacity, adhering strongly to the vessel containing it. So tenacious is it, that the vessel may be turned upside down without the sputa becoming detached from the sides. This rusty-coloured sputa consists of mucus intimately mixed with blood—not streaked with it, as in bronchitis, but thoroughly mixed and amalgamated with it—so that it acquires a yellowish, or reddish-yellow, or even a red colour, according to the quantity of the blood. If the disease be not very intense, the expectoration never attains the degree of viscidity or the depth of colour above referred to; but though still tenacious and adherent to the sides of the vessel, moves from one part to another as the vessel is tilted. If the disease progresses to a favourable termination, the sputa become more abundant, less adhesive, and less highly coloured, passing through the various shades of orange, until at length they become greenish or whitish, and resemble the expectoration of ordinary catarrh. If the disease be hastening to a fatal termination, the expectoration becomes scanty, less tenacious, and of a darker or dullish-brown hue, resembling the juice of prunes. If the type of inflammation be typhoid, or adynamic, or connected with tubercles in the lungs, the mucus may be tinged, or even streaked with blood; or it may consist throughout of nearly colourless, stringy, and more or less frothy mucus (Fuller *On Diseases of the Chest*).

Sputa Typical of Gangrene of the Lung, at first of a muco-purulent character, sometimes tinged with blood, begins to emit a very disagreeable odour; and as soon as a free communication is established between the air-passages and the sloughing tissue

of the lung, they not only acquire an intensely fetid gangrenous odour, but assume an appearance more or less characteristic of the disease. They lose their muco-purulent character, and become extremely liquid or sero-purulent, and of a dirty greenish or ash-grey colour. At the same time the breath acquires an offensive putrid odour, the pulse feeble and rapid, with evidence of great and increasing prostration, (FULLER, l. c.)

Sputa Typical of Acute Bronchitis appear, after a few days, as a thin, saltish, frothy mucus, sometimes streaked with blood. They increase in quantity, and soon become glairy, semi-transparent, and of a faintly yellowish colour. Subsequently they assume a greyish or greenish-yellow tint, and become opaque and viscid. If the attack is severe, they become muco-purulent, and in some instances may even lose their glairiness, presenting the character of thoroughly opaque mummillated sputa.

In *chronic bronchitis* the sputa may be of the following characters:—either (1.) The expectoration of a greyish, or greenish, or yellowish-white muco-purulent matter; or (2.) The expectoration being difficult, the sputa are comparatively scanty, consisting of stringy, tenacious mucus, of a greyish or yellowish-white colour, occasionally streaked with blood; at another time, expectoration being easy, the sputa are more copious, muco-purulent in character, of a yellowish-green colour, having a faint unpleasant odour; at another time the sputa are profuse, almost wholly purulent, of a nauseous and sometimes a fetid odour, usually running together into one mass, but often remaining separated, and forming distinct mummillated masses; or (3.) There is a profuse expectoration, sometimes to the extent of half a pint in an hour, of a thin, watery, ropy fluid, which varies in opacity, but is usually somewhat transparent, resembling gum-water (FULLER).

Sputa Typical of Plastic Bronchitis consist of ordinary bronchitic sputa, or blood-tinged mucus, with fragments of white fibrinous matter, or white fibrinous casts of the bronchi, which are ejected during violent paroxysms of cough. These concrete masses vary from mere fragments to large pieces of from *one* to *four* inches in length, and may be either tubular or solid, their ejection being preceded and often accompanied by spitting of fluid blood. These casts consist of concentric laminæ, found at different periods in successive layers, and consist of amorphous granular matter intermixed with mucus-corpuscles, compound granular cells, oil-

globules, and ovoid cells containing dark colouring matter, such as exists in ordinary bronchial mucus (FULLER).

Sputa Typical of Acute Phthisis consist—(1.) Of frothy mucus, often speckled with blood; and when the tubercle softens, the sputa become muco-purulent or purulent; or (2.) The sputa may be scanty, consisting of little more than frothy mucus; (3.) Expectoration, at first scanty, thin, colourless, and transparent, somewhat resembling saliva or gum-water, of a greyish colour, and more or less frothy. After a time the thin colourless sputa lose some of their transparency, and are seen to contain specks of opaque matter, which gradually subside and form a deposit resembling the sediment in barley-water; or they remain suspended by the more ropy part of the secretion, and float in the transparent mucous fluid in the form of striæ. Gradually becoming less aërated, they become more glairy and more tenacious, lose their pearly-grey colour, and are seen to be mixed with specks or streaks of an opaque white, or buff colour, and not unfrequently with specks or streaks of blood. (4.) As the malady progresses, the characters of the sputa change again. They become opaque, of a whitish or yellowish hue, and are coughed up in more distinct and homogeneous masses. Sometimes they form rugged pellets of a yellowish-white colour, resembling boiled rice, which sink or partially float in a colourless semi-transparent, ropy, non-aërated mucous fluid; or, accompanied by little mucous fluid, the sputa form large masses of a buff or yellowish-green colour, flocculent in appearance, but perfectly smooth in outline, which do not coalesce, but remain distinct and separate from each other if expectorated into a vessel of water.

All these forms of sputa occasionally occur in *chronic bronchitis* as well as in *phthisis* (FULLER).

Cough.—The severity, the frequency, or paroxysmal nature of the cough must be ascertained; also the circumstances which excite it most; and whether it is attended with pain, or followed by expectoration or vomiting. The ease or difficulty of the expectoration must be noted, and whether it is accompanied or not by pain. The quantity of the sputa ought to be measured in the day and night, the form of the masses spat up, their transparency or opacity, colour and viscidity, tenacity or adhesive property. The special characters of the sputa ought in every case to be closely observed, noting particularly its thin, serous, or frothy character;

whether it contains any membranous or concrete exudation-masses or blood; and it should be examined microscopically.

Pain.—The exact locality of pain in the chest should be ascertained, its severity, and the direction it tends to take. What particular circumstances aggravate it, and the effects of breathing, coughing, pressure, and postures should be ascertained.

Palpitation.—When *palpitation* occurs, its severity ought to be estimated by laying the hand over the region of the heart of the patient. It is desirable to ascertain its constancy; the circumstances which aggravate its existence or produce it—such as the influence of exertion going up a hill or up stairs, and the influence of mental emotion (*What to Observe*, pp. 39-44).

In the following table the more prominent characters of the palpitation which depends upon organic disease of the heart are contrasted with those of palpitation arising independent of disease of this organ (BELLINGHAM, *op. cit.*, p. 172):—

PALPITATION DEPENDING UPON ORGANIC DISEASE OF THE HEART.	PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART.
1. More common in the male than the female.	1. More common in the female than the male.
2. Palpitation usually comes on slowly and gradually.	2. Palpitation usually sets in suddenly.
3. Palpitation constant, though more marked at one period than at another.	3. Palpitation not constant, having perfect intermissions.
4. Impulse usually stronger than natural: sometimes remarkably increased, heaving, and prolonged; at others irregular and unequal.	4. Impulse neither heaving nor prolonged; often abrupt, knocking, and circumscribed, and accompanied by a fluttering sensation in the præcordial region or epigastrium.
5. Percussion elicits a dull sound over an increased surface, and the degree of dulness is greater than natural.	5. Extent of surface in the region of the heart, which yields naturally a dull sound on percussion, not increased.
6. Palpitation often accompanied by the auscultatory signs of diseased valves.	6. Auscultatory signs of diseased valves absent; bruit de soufflet often present in the large arteries, and a continuous murmur in the veins.
7. Rhythm of the heart regular, irregular, or intermittent; its action not necessarily quickened.	7. Rhythm of heart usually regular, sometimes intermittent; its action generally more rapid than natural.
8. Palpitation often not much complained of by the patient, occasionally attended by severe pain, extending to the left shoulder and arm.	8. Palpitation often much complained of by the patient; readily induced by mental emotion; and frequently accompanied by pain in the left side.
9. Lips and cheeks often livid; countenance congested; anasarca of lower extremities common.	9. Lips and cheeks never livid; countenance often chlorotic; anasarca absent, except in extreme cases.

PALPITATION DEPENDING UPON ORGANIC DISEASE OF THE HEART.	PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART.
10. Palpitation increased by exercise, by stimulants, and tonics, &c.; relieved by rest, and frequently, also, by local or general bleeding, and an antiphlogistic regimen.	10. Palpitation increased by sedentary occupations; by local and general bleeding, &c.; relieved by moderate exercise, and by stimulants or tonics, particularly the preparations of iron.

Expression of the Countenance in Thoracic Disease.—The countenance is often expressive of heart disease. In acute inflammatory affections of the lining or investing membrane it acquires an anxious and depressed character. An elevation and depression of the *alæ nasi* or *nares* are commonly observed with the respiratory acts; and the occurrence, in children, of these phenomena indicates a greater amount of disease than the general symptoms would lead us to suspect. In chronic cases, when the circulation is impeded, the expression of the countenance becomes almost pathognomonic. The venous system becomes congested, the face becomes bloated and dusky, the eyelids puffed, the eyes staring, the conjunctivæ suffused, the lips and cheeks purple, respiration laborious, the air-passages loaded with mucus, and the jugular veins distended or pulsatile.

CHAPTER IX.

DISEASES DURING WHICH LESIONS TEND TO BE LOCALIZED WITHIN OR ABOUT THE THORAX, ITS VISCERA, AND VESSELS THEREWITH CONNECTED.

Section I.—Diseases of the Walls of the Thorax.

In connection with thoracic pathology there are especially three affections which derive their interest, or rather importance, from the possibility of their being taken as evidence of pulmonary or cardiac disease. These are,—*pleurodynia* or *intercostal rheumatism, intercostal neuralgia,* and *thoracic myalgia.*

PLEURODYNIA—Syn., INTERCOSTAL RHEUMATISM.

Definition.—*Pain more or less acute, sometimes of agonizing severity, due to rheumatism of the walls of the chest, affecting especially their muscular and fibrous textures. The pain is most*

common on the left side, in the infra-axillary and infra-mammary regions. It is increased by deep inspiration, by coughing, by movements of the trunk and even of the arm, by decumbency on the affected side, and by pressure both on the ribs and in the intercostal spaces (WALSHE).

Pathology and Diagnosis.—The constitutional diathesis associated with rheumatism co-exists with this affection; and when the pain co-exists with acute articular rheumatism, the affection is associated with fever. It simulates the congestive or dry stage of pleurisy, and if febrile phenomena co-exist, the diagnosis becomes extremely difficult; and therefore a positive opinion should be withheld until, a certain number of hours having elapsed, the presence or absence of the characteristic friction-sound shall have established the nature of the case. If the pain extends very low down, it may simulate *peritonitis;* but the absence of rigors, of vomiting, of febrile phenomena, of the anxious expression of countenance characteristic of that disease, will always distinguish pleurodynia from peritonitis (WALSHE).

Treatment.—Friction with anodyne and stimulant liniments; the warm, vapour, or hot-air baths; rest; with colchicum in small, and bicarbonate of potash in free and oft-repeated doses, are the means which the pathology of the disease suggests as likely to effect a cure (WALSHE).

INTERCOSTAL NEURALGIA.

Definition.—*Pains intercostal, occurring paroxysmally, following the course of the affected nerves, usually involving the nerves of the thorax from the sixth to the ninth; more common on the left than the right side. The pain sometimes passes directly backwards from the edge of the sternum to the vertebral groove, and is frequently accompanied with pain in the branches of the brachio-cephalic plexus, sometimes in the gastric filaments of the vagus. In the intervals of the sharper pangs, numbness, coldness, and formication are occasionally felt* (WALSHE).

Pathology and Diagnosis.—This form of neuralgia is usually associated with hysteria and anæmia, especially in women weakened by *menorrhagia* or *leucorrhœa* (see Neuralgia, p. 507). It is frequently associated with phthisis. Three tender points in the course of the affected nerves indicate the true nature of the disease. These painful points are to be detected by pressure—one in the vertebral

groove, another about the axillary region, a third in front towards the terminal ramusculi of the nerves. There is sometimes general cutaneous hyperæsthesia in the affected region; and gradual but firm pressure over a hard surface usually gives relief. Months may elapse before a cure is effected; and the anæmic form is the most easily got rid of.

Treatment.—If local tenderness is extreme, leeches may afford relief, followed by a repetition of small blisters. A minor amount of pain, recurring from time to time, is best relieved by the endermic use of *morphia*, or by the inunction of ointments containing *belladonna* or *aconite*, or their *alkaloids*. *Quinine*, *arsenic*, and *iron* may be advisable in some cases from time to time (WALSHE).

THORACIC MYALGIA.

Definition.—*A painful affection, having its seat in the fleshy parts of the muscles of the chest walls, especially both pectorals, accompanied with exaggeration of the "muscular sense." It may be wholly subjective, or it may be a genuine hyperæsthesia excited by pressure and manipulation of various kinds* (WALSHE).

Treatment.—Rest, where the myalgia arises from over-work; while in the idiopathic variety, tonics, antispasmodics, and local anodyne applications, followed by stimulating douches, will generally effect a cure.

SECTION II.—DISEASES OF THE HEART AND ITS MEMBRANES.

Of Organic Diseases of the Heart.—Our knowledge of the anatomy as well as the pathology of the heart and large blood-vessels may be said to begin with Harvey; but the subject can hardly be said to have taken a scientific form till the beginning of the present century, when the work of Corvisart appeared, followed by those of Burns in England, of Testa in Italy, of Kreysig in Germany, and by the works of Bertin, and more especially of Laennec, Bouillaud, Senac, and Collin in France. A large school has since been formed in Europe by the labours of these eminent pathologists.

Nineteen years ago Dr. Williams and Dr. Hope, in their respective treatises, showed how it was possible to make an

accurate and minute diagnosis in almost every case of cardiac diseases; and the subsequent labours of Stokes, Graves, Bellingham, Forbes, Walshe, Davis, Sibson, Latham, Fuller, and Gairdner, in this country, have brought the pathology of diseases of the heart and lungs to their present advanced state of perfection.

The lesions of the heart are comprehended under diseases of the *Pericardium, Endocardium, Muscular Structure of the Heart* and of the *Blood-vessels.*

PERICARDITIS.

LATIN EQ., *Pericarditis;* FRENCH EQ., *Péricardite;* GERMAN EQ., *Pericarditis* —Syn., *Entzüngdung des Herzbeutels;* ITALIAN EQ., *Pericarditide.*

Definition.—*An inflammation of the fibro-serous membrane containing the heart, and investing it on its external aspect.*

Pathology and Morbid Anatomy.—The pericardium, like other serous membranes, is liable to inflammation, tending to the effusion of a serous fluid, diffused over a large extent of surface, and which sometimes becomes purulent—*suppurative pericarditis.* The morbid action may be of such a kind that the serous fluid coagulates upon the opposed surfaces, becomes vascular, and glues them together. These different forms of morbid action may co-exist, so that different parts of the pericardial surfaces may present very different appearances when examined after death. The inflammation may be acute or chronic, and attended with various productive results of the inflammation.

If the patient dies from acute pericarditis, the inflamed portion is of a bright rose colour. This redness is, in the first instance, caused by the increased vascularity of the subjacent areolar and fibrous tissue; but as the disease advances, red blood penetrates the serous membrane, and punctuates the exudation with a number of dots, which become confluent, and form patches that extend till perhaps the whole membrane is one bright scarlet colour. Besides being red, the membrane is thickened, first from coagulable lymph upon its surface, and then from the incorporation of interstitial growth with the sub-serous tissue. It is now opaque, white, thickened, and readily torn from its attachments; but the new membrane forms so complete a covering to the heart that the pericardial sac has to be forcibly opened up, so as to expose the organ within.

Dropsy of the Pericardium.—The inflammation may terminate by resolution; but more commonly serous effusion results, besides the solid lymph, the quantity of serum effused varying from a few ounces to a few pints. The solid lymph is then often found in shreds. Louis has given one case in which it amounted to four pounds, and Corvisart another in which the quantity was still more considerable. Walshe records sixty ounces — and when such great effusions occur, the diaphragm is depressed below the end of the sternum, by the great amount of fluid contained in the sack of the pericardium.

The adhesive results of inflammation often co-exist with the preceding inflammation, and lymph is generally formed in much greater quantity than from any other serous membrane. The lymph may be only in such quantity as to render the serum turbid, or so extremely loose in its texture as to float in it. More commonly, however, it is disposed as a membrane, often covering both surfaces of the pericardium, and especially that covering the heart, and measuring from two to several lines in thickness. This mass, when considerable, presents a remarkably irregular appearance, which has been compared to the stomach of a calf, to a portion of a honeycomb, or to two opposed surfaces united by grease and then forcibly separated. If the patient dies in the acute stage, this membrane is found only slightly coherent, and very rarely exhibits any trace of organization.

Suppurative Pericarditis. — When acute inflammation results in an accumulation of pus in the pericardium (which it does slowly), it is generally associated with a constitutional cause, and the pus may be of a laudable healthy character, though sometimes of a greenish hue. The quantity may be only a few ounces, or so abundant as to fill the pericardium.

Hæmorrhagic Pericarditis.—In cases where there is a disposition to purpura or scurvy, or during the progress of some malignant febrile disease, it occasionally happens that blood as well as serum or pus is effused within the sac. The source of the blood is from the new-formed vessels, which, being yet tender, give way; or it may be from the highly congested vessels of the serous membrane, and the softened tissues which prevail in scorbutus. The lymph of such cases is of a spongy red colour and shaggy appearance, exactly similar to that which extends from the large intestine into the small gut in cases of scorbutic dysentery.

Tubercular Pericarditis. — In the scrofulous cachexia, where

vomicæ of the lungs exist, pericarditis with minute tubercle on the pericardium may be also found. It is a common form of pericarditis in the army.

Adherent Pericardium.—*In the chronic forms of pericarditis* all these morbid states may be observed; and when lymph has been effused, it is then commonly found organized, so that the pericardium is often partially or universally adherent all over the heart. In some instances the lymph effused, instead of forming adhesions, becomes converted into cartilaginous and even osseous patches, which are readily detached from the surface of the heart by the scalpel.

The acute forms of pericarditis generally involve the muscular walls of the heart to a greater or less extent. On cutting through them the muscular substance is seen for a greater or less depth, of a deeper colour than usual, and the cohesion of the tissue is also impaired, the finger readily passing through it.

General Symptoms.—The symptoms of pericarditis vary much as regards their expression. In some instances they are most insidious in their approach; in others they appear to be violent and unmistakable from the commencement. In this disease rheumatism has been found to assume a new and formidable aspect; and rheumatic pericarditis is generally attended with more violent symptoms than non-rheumatic pericarditis. The most marked characteristic of rheumatic pericarditis is pain more or less severe in the præcordial region; and from this point it radiates over the whole of the sternum, sometimes extending to the brachial plexus and down the left arm. This pain is accompanied by disturbance of the heart's action, a sensation of constriction over the whole chest, by urgent distress, and by an incapacity to take a long breath, or to cough. From these causes the patient is restless and anxious, and this anxious expression of the countenance is often peculiar and striking from the first. When acute pericarditis is not the result of rheumatism, the patient may suffer no pain, and the symptoms are often most obscure, general as well as physical. The countenance is pallid, and assumes an aspect of distress, and there is an incapacity or unwillingness on the part of the patient to lie on his left side. The pain in the region of the heart is increased by pressure upwards against the diaphragm; his pulse, varying from 90 to 110, full and strong, and often intermittent, or otherwise irregular;

and this state of things having lasted from three to four days to a week, the patient may die suddenly.

Before death, and often throughout the more severe periods of the disease, there is delirium of a peculiar kind—sometimes quiet, but often wild and furious. This delirium is peculiar, and has been noticed particularly by Drs. Watson, Burrows, and others.

Even when the disease is most unmixed, it has been mistaken for a continued fever, or for pleurisy; and being usually one of the first complications of an otherwise fatal disorder, its mortality is thus far in excess of rheumatic pericarditis, which proves fatal in about one out of every six cases (FULLER).

Pure idiopathic pericarditis is rarely witnessed; and it very rarely occurs as a severe or clinically important form of disease.

Dr. Stokes, in respect of morbid anatomy, arranges cases of pericarditis into three classes. In the first class are to be placed those in which there is a slight though general effusion of coagulable lymph. In the second, those in which there is superadded the secretion of serum in abundance, causing distension of the pericardial sac. In the third class are to be placed those cases in which signs of muscular excitement, if not of myocarditis, are added to the preceding conditions. These three classes are thus contrasted, in respect of their symptoms or diagnosis, in the following tabular arrangement, given by Dr. F. Sibson:—

First Form.	Second Form.	Third Form.
Absence of pain or local suffering frequent. No sign of muscular excitement, nor any special character of pulse. No increase of dulness over the heart.	The local and general symptoms more decided, though often very trifling. Irregular action of the heart and pulse, often very manifest in the advanced periods. Remarkable increase of dulness over the heart.	Local distress, often extreme, even at the outset. Tumultuous action of the heart. Irregularity of pulse. Dyspnœa, orthopnœa, œdematous swellings, syncope, death.

As the disease passes from the first to the last of these forms, there is a progressive increase in the violence of the inflammation, denoted in the second form by the occurrence of excessive serous effusion, and in the last by the altered and impaired condition of the muscular substance of the heart itself. Death tends to occur by syncope, induced by paralysis of the left ventricle. Rokitansky thus describes the influence of pericarditis on the heart-tissue:—Its muscular substance is paralyzed, being of a dirty brown or yellow colour, flabby, and easily torn—a condition

which speedily leads to passive dilatation of the heart, general cachexia, and dropsy.

The first stage of pericarditis, before exudation, is not discoverable by physical signs (STOKES). This period rarely lasts longer than thirty-six hours. To Dr. Stokes, in 1833, the science of Medicine owes the description of the most characteristic physical sign of pericarditis. He established that in pericarditis a double *frottement* or friction-sound exists. Others also about the same time had noticed such a sound, and had correctly interpreted its meaning. Bouillaud and Collin, on the Continent, and Drs. Watson, Latham, and Mayne, in this country, had all, independently of each other, perceived and appreciated the symptom—a circumstance which, as Dr. Watson remarks justly, gives to the symptom a greater amount of importance. This sound closely resembles a rasping murmur. It has been named a "*to and fro*" sound by Dr. Watson. It is apt to disappear gradually from below upwards with the increase of effusion, and to return with its decrease. It may disappear from the apex to the base with the progressive formation of firm adhesions. It is usually limited to the region of the heart, but changes its character and its seat from day to day. It is sometimes remarkably modified by local bleeding, passing from a loud rough sound to a soft bellows murmur—most rough and intense during inspiration. The hand applied over the cardiac region will sometimes detect a rubbing sensation, which ceases with the cessation of pericarditis.

In diagnosis, however, it is necessary to bear in mind that *friction-sound is not necessarily present in pericarditis.* During the progress of a case, friction-sound may be absent or it may be present for long periods—its presence or its absence bearing no appreciable relation to the intensity of the disease. The amount of fluid effusion has much to do with this. A really considerable effusion of fluid generally at first muffles, then renders barely audible, and finally removes the sound, the friction-sound becoming indistinct as the heart's sounds are gradually extinguished. Like the heart's sounds, the friction-sound continues audible longest, and is recovered soonest towards the base. When the lymph is rapidly condensed into firm granulations, and the parts of those granulations most exposed to attrition have become polished and rubbed away, so that the points gradually receding from each other present fewer and fewer points of con-

tact, then the friction-sound may subside, although no further effusion of fluid takes place (W. T. GAIRDNER).

Again, *the presence of friction-sound is not necessarily a proof of the existence of pericarditis.* There are permanent exocardial murmurs, probably associated with the well-known "*milk spots*" on the anterior aspect of the heart, as pointed out by Professor Gairdner. When it is remembered how frequently slight, short, and ill-defined murmurs, especially with the first sound of the heart, may be discovered, in persons otherwise healthy, about the left border of the sternum, at the level of the third and fourth intercostal spaces, or lower, the existence of "milk spots" may be their cause. *It is only when the murmur arises for the first time under observation, or when it accurately coincides with the development of symptoms, or where it corroborates and explains the symptoms, and the other physical signs already existing, in such a manner as to leave no doubt of its nature, that we are justified in assuming that a friction-murmur over the heart is pathognomonic of acute pericarditis.*

The *white* or *milk spot* is so often found in hearts which in other respects are perfectly healthy, that many pathologists doubt their morbid nature (BAILLIE, SŒMMERING, HODGKIN, J. REID). The anterior surface of the right ventricle is their most frequent seat. Occasionally these spots are observed upon the surface of the left ventricle, or upon the auricle, or upon the prominences of the coronary vessels. Their size varies from a fourpenny piece to a crown or larger. They are more common in adult than in early life; but they have been observed in the infant under three months old. They increase after the age of eighteen, apparently progressively with age. About 33 *per cent. post-mortem* examinations from the ages of eighteen to thirty-nine show such *white spots;* and about 71 *per cent.* from ages between forty and eighty. Baillie, Laennec, Louis, and Todd, state that these opaque patches can easily be dissected or peeled off from the visceral layer of the pericardium, leaving the membrane entire. Corvisart, on the other hand, says they are seated on the under surface of the membrane. There seems, however, to be two forms of the white spot—namely, (1.) A superficial, which may be peeled off; and (2.) A deeper spot, which cannot be so detached (BIZOT, PAGET, KING, HODGKIN).

A great difference of opinion prevails as to the cause of these

spots. Some may, no doubt, be due to previous pericarditis; but the weight of evidence seems to be in favour of attrition being their cause (HODGKIN, KING, JENNER, WILKS). The circumstances which seem to favour the development of this white spot are those which would increase the rubbing of the part against the pericardium applied to the anterior wall of the thorax. Those circumstances are—(1.) Dilated heart; (2.) Impeded action of the lungs (*a.*) from those diseases which, leading to augmented volume of the lungs, tend to press the heart forwards; and (*b.*) from continuous pressure upon the chest in an antero-posterior direction, commenced at an early age, before the epiphesis of the ribs and the pieces of the sternum are fully grown and united—as in young soldiers who, during great exertions, carry a loaded pack and wear cross-belts.

These pericardial or exocardial friction-sounds or murmurs may be, and are often, mistaken for endocardial murmurs (TAYLOR, STOKES, GRAVES, SKODA, SIBSON), and the distinction between exocardial and endocardial murmurs is not always easy, nor is it to be effected by the ear alone. The following distinguishing signs are condensed from Dr. Sibson's interesting review of the works of Stokes and Bellingham on diseases of the heart (*Medico-Chirurgical Review* for 1854).

The exocardial may be distinguished from the endocardial sounds by the nature and nearness of the exocardial sound—by its existence with diastole as well as systole—its limitation to the region of the heart—its non-existence over the great vessels—its variations over different parts of the heart—its rapid and frequent change in character, or its disappearance from day to day—its want of correspondence with the rhythm of the heart, while it seems to follow upon its movements (SKODA), or to precede and follow the impulse (WUNDERLICH)—its co-existence with tactile vibration, and where there is much effusion, with an extensive cone-shaped region of cardiac dulness. The apex of this dull region points to the top of the sternum, its broad base extending downwards to the right, and far to the left of the epigastrium. In addition to these signs, which, when they exist, serve to establish the existence of pericarditis, and separate pericardiac from endocardial murmurs, we have other very characteristic signs by which to distinguish every case of pericarditis, especially in the earlier stages. By the aid of pressure, as first demonstrated by Dr. F.

Sibson, applied gently over the region of the heart, we have a test decisive as to the cause of these sounds, when we are in doubt as to whether it is endocardial or exocardial. If the noise is that of a valve-murmur, pressure from without does not increase or modify it, except in some anæmic persons, over the aorta. If, on the other hand, it is that of a friction murmur, soft and bellows-like, of exocardial origin, pressure intensifies the noise, and converts the sound into a rustle or rub. By pressing gently on the costal cartilage or end of the sternum with the stethoscope, the intermediate fluid is displaced, and the roughened surfaces are brought into contact. This method of diagnosis is most valuable, especially in the early stages, when it is of real importance to arrive at a correct diagnosis (*Provincial Med. Trans.*, vol. xii., p. 540).

"A really refined and intellectual diagnosis," and not one founded on the mere *aural* recognition of acoustic characters, is necessary to guard against serious mistakes. In the majority of cases where the friction-sound of pericarditis is recognized, it is known to be such by the circumstances in which it occurs rather than by the mere character of the sound itself (STOKES, WALSHE, FULLER, GAIRDNER). Increased extent of dulness in percussion, and marked prominence over the cardiac region, are also two characteristic signs.

When extensive effusion takes place, the heart is pushed upwards to the fourth, third, or second intercostal spaces; consequently the seat of the heart's impulse, of the rubbing sounds, and of tactile vibrations, all are correspondingly raised (SIBSON, LATHAM, WALSHE).

A valuable distinctive sign of pericardial effusion, when contrasted with pleuritic effusion, is, that when the left side is dull in front and resonant behind, it is a pericardial, and not a pleuritic effusion (STOKES). Any large increase of fluid at once betrays itself, especially in the young, by the protrusion of the left cartilages and ribs, the widening of their interspaces, prominence of the ensiform cartilage, and, in some extreme cases, by an epigastric fulness or even tumor. When the fluid increases, the pulse becomes feebler, and more disposed to falter and to flutter. It becomes irregular and excited; and often the patient is so fixed in one position that he fears to move, lest he may aggravate by exertion the dyspnœa and action of the heart from which he suffers so intensely. The

jugular veins not unfrequently become distended, and this distension does not lessen during inspiration when the effusion is great. A significant sign is thus furnished of the greatness of the obstruction which exists to the thoracic circulation. Œdema and great coldness of the extremities are also apt to supervene with such a state of things. When, however, the products of inflammation become solid, and little serum remains, the pericardium, by the opposed serous surfaces, becomes attached to the heart throughout, and the pulse then resumes its force and regularity; and, if the patient survives, this adhesion remains for life.

The occurrence of acute pericarditis apart from vital phenomena or symptoms is not now believed in, and among the most characteristic general symptoms is the occurrence of a dry short cough, not explained by any morbid state of the lung.

Prognosis.—While the consequence of pericarditis "is sometimes, though rarely, the speedy extinction of life, in nineteen cases out of twenty the disorder proves fatal at a remote period, destroying the subject of it more slowly, indeed, but almost as surely." Such is the opinion of Dr. Watson; and again he writes, "I am certain it is a fertile, but often unsuspected source of chronic disease of the muscular substance of the heart, and of its consequences, asthma, dropsy, sudden death." Sometimes death occurs suddenly when least expected; and the mode of death tends to be by asthenia. A fatal termination may occur, although rarely, in less than forty-eight or thirty hours. In ordinary cases which progress favourably, the disease generally begins to yield in the course of a week or ten days, and sometimes sooner under active treatment.

It is not now, however, generally believed that when the disease terminates by adhesion, dangerous or fatal lesions of the heart tend to supervene, as Dr. Hope believed. It is consistent with the experience of Drs. Stokes, Sibson, Wood, of Pennsylvania, W. T. Gairdner, Smith, and other observers, that hypertrophy and dilatation of the heart do not commonly follow on pericardiac adhesion. Dr. Watson, however, with many others, regards adhesion of the pericardium as a suspicious prognostic of future evil—"that other structural changes will soon or slowly develop themselves; and first render life burdensome and full of suffering, and then consign the patient to an earlier grave than might else have awaited him."

Among the patients of a general hospital, from 6 to 8 *per cent.* die of pericarditis, while a proportion, varying from 3 to 8 *per cent.*, have had the disease at a previous period and recovered from it (W. T. GAIRDNER).

Cause.—Pericarditis is generally the local manifestation of a disease which pervades the system. There is scarcely a form of constitutional malady which may not be accompanied by *pericarditis;* but those with which it is most apt to be associated are *rheumatic fever, Bright's disease, pyœmia, pneumonia, tubercular vomicæ, pleurisy, small-pox, scarlatina, scurvy.* These diseases are mentioned in the order of the frequency with which they give rise to pericarditis, as consistent with the observations of Drs. Taylor, T. K. Chambers, A. W. Barclay, and Dr. Fuller. In cases of *acute rheumatism,* pericarditis occurs once in every 5·97 cases (FULLER).

The muscular substance of the heart sometimes suffers in cases of pyæmia consequent on the vitiated state of the arterial blood, and pericarditis has been known to arise in this way (Kirkes, *Med. Times and Gazette,* October 25, 1862, p. 432). Such cases have usually been described as cases of carditis, or acute inflammation of the muscular substance of the heart depositing pus. Dr. Kirkes, however, believes that the muscular tissue of the heart is not primarily at fault, but, in common with other parts of the body, it is by accident the seat of secondary formations in consequence of contaminated blood; and the attendant pericarditis is an accident of the proximity of some of the deposits to the surface, and it may be of their bursting into the cavity, just as an ulcer in the wall of the intestine may burst into the *peritoneum* and cause *peritonitis.* It has also occurred by extension of other diseases from neighbouring organs—*e. g.*, cancer of œsophagus, and the like (J. W. BEGBIE). (For more details regarding pericarditis, the student is recommended to study the excellent treatises of Dr. Fuller *On Rheumatism* and *On Diseases of the Chest;* also Walshe and Stokes *On Diseases of the Heart*).

Treatment.—The results of recorded treatment are unsatisfactory in the highest degree. Dr. Watson thus writes, that "in a large proportion of the cases, whether they be treated well or ill, or not treated at all, the patients will seem to recover." Although blood-letting and calomel have hitherto been regarded as two most efficient agents in accomplishing the indications of cure, yet

Taylor's cases show that pericarditis may come on during salivation. Dr. Parkes informs me that he has seen this occur two or three times. It is, then, very doubtful if calomel ever does any good in pericarditis, notwithstanding that its use, so as to affect the gums, is advised by Watson, Graves, Stokes, and Fuller. Professor W. T. Gairdner, Drs. John Taylor, of Huddersfield (*Med. Times and Gazette*, 1849), and J. Risdon Bennett, of London (*Lancet*, Dec. 6, 1851), are all unfavourable to the use of mercury. In all the *constitutional diseases* in which pericarditis occurs as a complication, mercury is certainly contra-indicated. In rheumatism and in Bright's disease, which furnish by far the larger number of cases of pericarditis, mercury is decidedly objectionable; and it is now well known to be productive of most injurious consequences in Bright's disease. Its use tends to weaken the heart and the system at large, and thus proves a source of additional irritation to the cardiac tissue, by undue re-action on the system.

Dr. Fuller, however, gives his opinion very strongly in favour of mercury as a remedy in the acute and sthenic form of pericarditis, as it presents itself in persons of a strong and healthy constitution. "No such case," he says, "can be treated safely without its administration." Such idiopathic cases are indeed rare, the causes which induce pericarditis being for the most part constitutional.

"A single bleeding from the arm," writes Dr. Stokes, "appears on the whole justifiable, but its repetition will be a matter for careful consideration." It is a remedy which has been clearly shown by the late Dr. Taylor to shorten the duration of pericarditis, and to do so the more effectually the earlier it is performed. (*Medical Times and Gazette*, 1851). The treatment by bleeding may also be further carried out by leeches over the præcordial region, or by cupping there. In cases of rheumatism, Drs. Stokes and Gairdner hold that "it cannot be too strongly impressed upon the mind that, valuable as the discovery of the signs of an inflamed pericardium may be, it is not for these alone that he is to look, but rather for the indications of excitement of the heart, whether attended or not by the signs of exocardial or endocardial disease." These indications may demand local depletion, "even though no friction-sound or valvular murmur whatever be present." Dr. Watson gives a most judicious opinion when he says,—

"I think there is a peculiar risk in frequently bleeding to syncope in this affection." Blood may be drawn from the arm till some effect upon the pulse is produced; but Dr. Watson gives the preference to repeated relays of leeches over the præcordial region, or to free cupping. Dr. W. T. Gairdner advises that when the pain is very marked, where it has strongly the characters of angina, leeches are to be applied in moderate numbers. From *four* to *six* leeches so applied, followed by fomentations, very commonly relieve the pain, and rapid improvement follows.

To judge as to the repetition of blood-letting, the force of the heart must be observed, as indicated by the pulse at the wrist, by the actual strength of its impulse, and by the character especially of the first sound. If the impulse continues vigorous, and the first sound is undiminished, blood-letting may be repeated by the further *local* abstraction of blood; but if the impulse has manifestly declined in force, while the first sound is lessened, great caution is required in the further abstraction of blood. Fomentations, sometimes plain and sometimes medicated with opium, friction with camphorated and iodureted liniments, and, in obstinate cases, the use of blisters, are the chief local remedies to be relied on besides leeches.

In the second stage of the disease, when liquid effusion distends the pericardium, some reliance may be put in treatment by a blister of a large size over the præcordia.

"Stimulants," writes Dr. Stokes, "are often imperatively called for. The weakened heart requires to be supported and invigorated. This may be effected by the use of wine, and by the use of opium." If depletion has been excessive—if signs of muscular weakness supervene—if there be evidence that the heart, previous to the attack, was in a weakened state—if a tendency to collapse or to a typhoid state manifests itself—"we must give wine, quite irrespective of the physical conditions of the heart" (STOKES).

Opium, in doses of one grain (*i. e.*, stimulant doses) every third hour, seems "to expend itself solely on the disease," and its beneficial effects are seen to result when it does not produce contraction of the pupil, nor headache, hot skin, furred tongue, nor constipation (CORRIGAN, STOKES, GRAVES, SIBSON). Battley's solution is perhaps the most useful preparation.

There are two more important circumstances to be attended to,

as stated by Dr. Sibson—namely, that our treatment of pericarditis must depend upon the stage of the disease in which it is first discovered to exist; and it is important to know that pericarditis from acute rheumatism calls for a totally different line of treatment from that associated with Bright's disease, or diffused inflammation of a low type.

In the rheumatic form, colchicum, in the form of a draught, and the administration of alkaline remedies, are indicated by the constitutional state.

Paracentesis is recommended by Dr. Sibson in all those cases in which the effusion is so great as to cause alarming distress, orthopnœa, obstruction to the venous circulation, and serious interference with the heart's action. In such cases a fine exploring trochar and canula is to be introduced (not plunged), so as to make a valvular opening below the heart, either to the left of the xyphoid cartilage, or through the fifth intercostal space, close to its anterior extremity, and the fluid drawn off by means of a syringe (Sibson, in *Med.-Chir. Review*, July, 1854).

The general principles of safe treatment are summed up by Dr. W. T. Gairdner as follows:—(1.) To make large allowance for the insignificant and spontaneously healing class of cases revealed more by physical signs than by symptoms, and to regard them as demanding little active treatment; (2.) To consider rheumatic pericarditis in general as a disease susceptible, to a great extent, of cure under palliative local remedies and fitting constitutional treatment; (3.) To hold the general treatment as subordinate to the constitutional treatment of the disease with which the pericarditis is associated.

DISEASES OF THE ENDOCARDIUM—ENDOCARDITIS.

LATIN EQ., *Endocarditis;* FRENCH EQ., *Endocardite;* GERMAN EQ., *Endocarditis;* ITALIAN EQ., *Endocarditide.*

Definition.—*An inflammation of the lining serous membrane, or endocardium, covering the valves and lining the chambers of the heart.*

Pathology and Morbid Anatomy.—By a species of preference the coverings of the orifices and valvular structures of the heart are by far the most frequent seat of lesion in the internal inflamma-

tion of that organ. The frequency with which these parts suffer may in some measure be explained, if their minute anatomical and histological relations are connected with the morbid states of similar tissue. The peculiarity of the minute structure of these parts, as influencing the arrangement assumed in the first instance by morbid deposits, seems to have been first definitely stated and illustrated by Dr. Watson, (Lect. lxi., p. 275, 3d edit.) There is found to exist enclosed between the reduplications of the endocardium a quantity of fibrous tissue. An increase in its amount takes place at the centre of each aortic valve, forming the *corpora Arantii*, and at each of the extremities or angles of the segments. The minute exudations which are formed as the result of the inflammatory process in the endocardium may be seen to arrange themselves in double festoons, suspended as if from the *corpus Arantii*, often in a row, like a string of beads, along the line of union of the thick portion of the valve, with the inner convex margin of its two thinner crescentic portions. The repeated attrition of the opposed surfaces of the valves pushes aside the exudation as fast as it is deposited, and while yet plastic, from the thin crescentic portions of each valve, and so heaps it up along those boundary lines of contact; "just as a thin layer of butter on a board would be displaced, and heaped up in a little curvilinear ridge, by the pressure of one's thumb."

While, doubtless, the structure of the serous membrane is the same throughout the heart, yet its mixture with white fibrous and elastic tissue, in great abundance at the valvular reduplications, renders it prone to disease, especially in those constitutional states of the system in which the fibrous textures are more particularly involved, as in rheumatism. These are the parts also on which the great tear and wear on the action of the heart is expended, and thus they are probably the first to suffer, owing to the mutual friction of the valvular edges upon each other.

Diffuse inflammation of the endocardial membrane has been known to follow the application of a ligature round an artery. The morbid appearances of its inflammation are similar to those in other serous textures—namely, a silvery opacity, and more or less thickening of its tissue. Inflammatory lymph is often found strongly adherent to the valves, as already described, and forming fringe-like or fibrinous warty growths, or excrescences, as they are termed. By its agency the segments or lappets of the valves

become variously distorted in shape, or soldered together, and insufficient to perform their functions. An orifice, naturally large, may thus be reduced to the condition of a mere slit, or to the diameter of a goose quill. One segment of the aortic valve may, for example, be turned up and bound to the aorta, or it may be turned down and bound to the inner surface of the heart, or it may be curled up like a shell. The lappets of the mitral valve may be similarly altered.

The prolonged existence of the inflammatory state ultimately thickens and hardens, by interstitial deposit, the tissue enclosed between the folds of the serous membrane constituting the valves, so that their action is much impaired. These changes may be limited to the fibrous zone which forms the base of the valves, surrounding the aortic orifice with a sort of collar, contracting its diameter, as well as impeding the play of the valves. In other cases the thickening may affect the free edge, or the central portion of the valve. The most remarkable circumstance, however, connected with chronic adhesive inflammation of the left side of the heart, is the excessive tendency which the valves have beyond all other serous tissues to become cartilaginous or ossified. This transformation commences in the substance of the serous tissue, but more commonly in the tissue connecting the duplicature of the valvular fold. This ossific deposition is not necessarily preceded by a cartilaginous formation, but is most frequently an original abnormal secretion, often containing a good deal of earthy matter. In all the instances in which I have examined such deposits, they did not exhibit the histological appearances of true bone-tissue. It is deposited in various forms: sometimes in layers, at others in points, and at others in large masses, in knobs or pyramids, occasionally acquiring a size as large as a pigeon's egg. Sometimes the tendons, or the *chordæ tendineæ* attached to the mitral valve, participate in these indurations, and Corvisart met with one entirely ossified. The irritation of these deposits often leads to their destruction, and the whole exudation softening and breaking down, may mingle with the current of the blood, and produce results, to be noticed, of a most serious description.

Dr. Latham and Dr. Hope were of opinion that endocarditis is more frequent than pericarditis. Dr. Stokes, Dr. Sibson, and others entertain a different opinion, consistent with the evidence

of *post-mortem* inquiries, as recorded by Drs. T. K. Chambers, A. W. Barclay, and Taylor.

The tendency of endocarditis is—

1. To produce those affections of the heart which are also described respectively as "*valvular disease of the heart*"—"*hypertrophy*," and "*dilatation*"—morbid conditions more or less simple or combined.

2. Associated as it often is with pericarditis, and acknowledging rheumatism as a most frequent exciting cause, we have the *muscular* substance of the heart itself sometimes affected, constituting what Dr. Watson terms "*rheumatic carditis.*"

Symptoms and Results of Endocarditis.—A more extensive, forcible, and abrupt impulse of the heart than natural, combined with endocardial murmurs, of a soft low pitch tone and blowing sound, combined with a febrile state of the system, and cardiac uneasiness, suggest the probability of endocarditis. A careful study of the development, order of occurrence, and combination of the general symptoms and physical signs, can alone convert that probability into a certainty.

1. **General Symptoms.**—The patient is observed to prefer to lie on his back (dorsal decubitus), and he may perhaps incline to toss about with his arms. Pyrexia may prevail of a specific kind, as when rheumatism, Bright's disease, or typhus fever is present, or it may be idiopathic inflammatory fever, associated with the endocarditis. So long as the cardiac orifices are not seriously obstructed, and no obstruction exists in the lungs from pneumonia or bronchitis, no special sensation of dyspnœa is complained of. The pulse ranges in frequency between 80 and 120, and it has been stated by Dr. Taylor even to lose in frequency at the outset of the affection. More or less headache may prevail.

2. **Local Symptoms.**—Discomfort and uneasiness at the heart are most common symptoms, and more or less palpitation may be present.

The extent and power of the impulse of the heart ought now to be examined carefully and repeatedly, and the conditions which tend to subdue or to aggravate these phenomena ought, if possible, to be ascertained. The areas of the heart's dulness, both superficial and deep-seated, undergo increase (BOUILLAUD, WALSHE).

The murmurs which accompany endocarditis purely acute are thus arranged in the order of their frequency by Dr. Walshe:—(1.) Aortic obstructive; (2.) Mitral regurgitant; (3.) Aortic regurgitant; (4.) Aortic obstructive and mitral regurgitant together. (The student is referred back to page 565, *et seq.*, for the account of the sites of maximum intensity where these murmurs may be listened to.)

The comparative frequency of aortic and mitral valve disease has been determined with more accuracy by the results of the combined observations of Drs. Barclay, Chambers, and Ormerod, as tabulated by Dr. Sibson. From these records it is seen that the *mitral valves* are more subject to disease than the *aortic;* that the disease is often limited to one set of valves, but that it is more often common to both valves than limited to either. When associated with acute rheumatism, the disease affects both valves in the greater number of cases; and the *mitral* more frequently than the *aortic.* In the young, who are subject to acute rheumatism, disease of the *mitral* valve, and in the old, who are subject to atheroma, disease of the *aortic* valve predominates. In the more severe cases in which the valve disease is itself the cause of death, the *mitral* valve disease is shown to be the most prone to go on to a fatal issue (*Med.-Chir. Review*, Oct., 1854, p. 431).

As there is no difference in character between the murmur of endocarditis and that which attends established valvular disease, it is necessary, in order to appreciate the existence of endocarditis more certainly, that the *murmur should be developed under observation at the early period of an acute attack* (WALSHE): and if a mitral or aortic murmur supervene while a case of acute rheumatism is being watched, especially if there be congestion and an expression of anxiety in the face, with distress in the region of the heart, *not caused by pericarditis,* there is a strong probability of endocarditis (SIBSON). But the symptoms are often exceedingly insidious in their origin and progress, and the disease is rarely simple, being generally combined with pericarditis; and, moreover, as the *general* constitutional symptoms of these two diseases do not differ, the detection of endocarditis, *per se,* is one of the most difficult in practice. Like pericarditis, it is often latent, as in rheumatic fever, and the practitioner is often surprised by his patient showing symptoms of valvular disease after an apparently

perfect recovery from fever (STOKES). A murmur, *per se*, is no sufficient evidence of endocarditis. Stokes, Sibson, and Graves have each of them recorded cases where *mitral*, and still more often *aortic* murmurs have been generated, when no valvular disease existed,—in cases of fever especially, in a case of fatty degeneration of the heart, and in a case of pericardial adhesion. These murmurs have also been recorded to exist during life, in cases in which no trace of valve disease was observed after death, by Drs. Barclay, Markham, Chambers, and W. T. Gairdner (Sibson, in *Med.-Chir. Review*, Oct., 1854).

As in pericarditis, it is important to recognize the *friction-sound* pathognomonic of its existence, apart from any endocardial murmur with which it might be confounded; so in endocarditis it is, if possible, still more important to detect endocardial murmurs when masked by pericarditis, for the grazing sounds of the latter disease may altogether mask those of the valve murmurs. The principles on which the diagnosis is to be effected are involved in the facts that friction-sounds of pericarditis are limited to the heart's region (STOKES); while, as Dr. Walshe so clearly describes, the sounds of the heart, and the murmurs which attend the lesions of its valves, are propagated in certain determinate directions; and while they are heard in maximum intensity at certain points, more or less defined, they may be detected by following the line of propagation at points beyond the mere limits or region of the heart itself. Upon these grounds data are furnished by which to distinguish the murmurs of endocarditis. (See *ante*, p. 565, *et seq.*)

For this purpose the murmurs of endocarditis must be looked for in suspicious cases from day to day *beyond the region of the heart;* and if a systolic mitral murmur is heard extending an inch and a half beyond the nipple, it is most probably due to mitral regurgitation (SIBSON). The detection of an aortic murmur with pericarditis is much more difficult, because the *friction-sound, frottement*, or "to and fro" sound, often mounts to the top of the sternum. The aortic murmurs are therefore to be listened to in the line of the natural propagations of the aortic sounds; and if an aortic murmur exists, it can only be distinguished in the neck, the best point for examination being just above the sternum, a little to the right, over the *innominata*. If, after listening to the first sound, the second sound be

observed to follow clearly and distinctly, the chances are that there is no affection of the aortic valves, even if there be a loud systolic murmur. If, however, the second sound be indistinct, inaudible, or prolonged, or be replaced by a diastolic murmur, aortic endocarditis may be suspected or detected (SIBSON). (The student is requested to contrast p. 565, *et seq.*, with these statements.)

As far as the immediate practical value of the information is concerned, it seems to be really unimportant where the exact seat of the murmur is. It is of no practical importance, for example, in the first instance, whether the murmur proceed from a "mitral, a tricuspid, or a semi-lunar valve, or whether it may be due to a contraction, or a dilatation, an ossification, a permanent patency, or a warty excrescence." The practical points to be determined, in the first instance, reduce themselves to two—namely, *First*, Do the murmurs proceed from an organic cause? *Second*, What is the vital and physical condition of the texture of the heart itself with which they are associated?

For subsequent practical purposes the limits of inquiry may also be very much circumscribed—namely, to the recognition of the occurrence of contraction or of dilatation of the orifice, because both of these conditions are attended with a permanently open state.

While, therefore, the occurrence of murmurs, and their nature, and the circumstances under which they are developed, are of the utmost importance to establish the existence of an endocarditis, the condition of the muscular substance of the heart must be the great guide in prognosis and treatment. The vital and mechanical state of the heart's cavities must be ascertained. The action of the heart must be carefully noticed at different times, as to whether its force and vigour is above or below the natural standard—whether it is liable to excitement from slight causes—and whether it tends to regular or irregular action, as regards rhythm or frequency of revolutions. The duration and origin of the disease must be ascertained; and how far the brain, lungs, or liver suffer from the mechanical or vital effects of the lesion (STOKES).

The symptoms, therefore, of acute endocarditis being detected, the immediate treatment of the disease must be proceeded with; and the physician requires also to look before him in anticipation

of the results which are likely to ensue if valvular lesion is established, such as hypertrophy and dilatation (which see).

Another class of symptoms and results is apt to be associated with endocarditis and valvular disease—as when products of inflammation are apt to poison or spoil the blood. Rigors, heat of skin, profuse perspiration recurring irregularly, dull, earthy-yellow discoloration of the skin (not of the conjunctivæ), diarrhœa more or less bilious, pinched, anxious countenance, intense prostration, and muttering delirium, are the symptoms which Dr. Walshe describes as announcing this untoward occurrence. Secondary deposits in the lungs, the liver, or the brain, are the records of its morbid anatomy. But, again, these secondary deposits may not be the direct result of existing endocarditis. Virchow, Kirkes, Simpson, and Rokitansky have shown how the fibrinous coagula, which have become permanently attached to the valves, constituting the vegetations upon them, may become worn away superficially, and taken into the blood in fine particles, thus leading to secondary coagula in the capillaries of the spleen and kidneys, to obliteration of these vessels, and in the capillaries of the brain leading to softening, and sometimes to sudden death. The great tendency to the formation of these coagula on the valves, in rheumatic pericarditic attacks, must be specially remembered when it is determined to abstract blood, as the slowness of the heart's action which may ensue greatly favours the tendency to coagulation of the blood and to the deposition of fibrinous deposits on the valves.

Causes of Endocarditis.—The inner membrane of the heart (exposed as it is to the action of many morbid poisons, and also to many substances which may be taken up by the absorbents and introduced into the circulation) is not found so frequently diseased as we might expect. Of all substances, however, alcohol has the most striking effects on this tissue. It is not only proved to be absorbed and actually to circulate in the blood, but there are few drunkards the inner membrane of whose heart and large vessels is not more or less diseased; so that alcohol probably acts as a specific poison on the endocardium. The morbid conditions associated with Bright's disease, with rheumatism and gout, and with syphilis, appear to act especially upon this tissue, and many who suffer from these diseases often ultimately die of some

form of endocarditis often extending along the lining membrane of the aorta.

Prognosis.—So far as the endocarditis is concerned, the immediate prognosis is very similar to that stated under pericarditis; but the future chances of life being prolonged depend upon the lesions which remain permanent. If valvular lesion is fully established, and remains persistent, hypertrophy is certain to follow, and the danger will be the greater in proportion as dilatation of the cavities of the heart predominates over hypertrophy. "Under all circumstances," writes Dr. Walshe, "dilatation is a most serious disease; and the danger increases directly as the excess of the capacity of the cavities over the thickness of their walls; directly, too, as the softness and flabbiness of the heart's tissue; directly, too, as the general deficiency of tone in the system and impoverishment of the blood. Once dropsy has supervened, life can with difficulty be prolonged by art beyond twelve or eighteen months. Dilatation of indubitable existence is not removable by *treatment*" (Walshe *On Diseases of the Heart and Lungs*, p. 487). If neither aortic lesion, hypertrophy, nor dilatation results, the tissue of the heart itself may be so impaired as to lead to softening, as in typhoid and typhus fevers, scurvy, or purpura. Fatty degeneration of the cardiac tissue is also a result to be apprehended.

The valuable medical reports of St. George's Hospital, London, prepared by Drs. A. W. Barclay and Rogers, contain the following statistics relative to the *per centage* of mortality *among hospital patients* from *diseases of the heart* during a period of six years—namely, from *pericarditis*, 34·8; from *endocarditis*, 9·19; from *hypertrophy*, 60·5; from *dilatation*, 52·1; from *valvular disease*, 24·5.

Treatment.—What has been written relative to the treatment of *pericarditis* applies equally to acute *endocarditis;* but the management of cases in which the patients suffer from the valvular lesions and their immediate consequences demands the adoption of various lines of treatment. From whatever cause, it is one of the most intractable diseases with which we are acquainted.

When endocarditis seems lapsing into the chronic stage, Dr. Walshe recommends the use of *iodide of potassium* and *liquor potassæ*, combined with bitter tonics.

(CHRONIC) VALVE DISEASE.

LATIN EQ., *Morbis valvarum;* FRENCH EQ., *Maladies des valvules;* GERMAN EQ., *Klappenkrankheiten;* ITALIAN EQ., *Malattie delle valvole.*

Definition.—*Lesions of the valves of the heart, or of its orifices, coming on for the most part insidiously, and which, persisting, induce obstruction or regurgitation, tending to hypertrophy and dilatation of the heart, with congestion of the pulmonary and systemic capillaries, œdema, anasarca, and dropsy.*

Pathology.—The term "chronic valvular disease" employed here is used in order to distinguish the chronic lesions of the valves of the heart from "chronic endocarditis;" for it has been already shown that the valves and orifices of the heart are often damaged by this affection. But chronic valvular disease does not always owe its origin to endocarditis. Such chronic valvular disease is often very insidiously established as a local expression of chronic Bright's disease, or chronic rheumatism, and in gout, as well as in such forms of constitutional derangement of the system as are associated with imperfect nutrition of the body. A very slow deposition of fibrin on the substance of the valves, or degeneration of their structures, is the usual lesion they present. The valves ultimately become thickened, opaque, and puckered, and may be rigid by the presence of atheromatous or calcareous matter. Yielding under pressure, they are apt to become thinner than natural, or they become perforated or cribriform—a condition which may also be referable to congenital deficiency. Sometimes they rupture, and present all the phenomena of ulceration, or their surfaces are irregular and their edges beaded by the deposit of fibrin from the blood. One segment of a valve may be found adherent to another, and in each of these cases the disease may be of such a nature as to obstruct the onward flow of blood, or to permit regurgitation through the diseased valves, when an obstructive or regurgitant murmur will be the result (see *ante*, under "Cardiac Murmurs"). The orifices of the heart may be the parts diseased, while the valves are sound. In such cases the orifices are usually roughened by calcareous or atheromatous matter, or they may be so much dilated that the valves are insufficient to shut them. The lesions to which valve disease may be referred are of the following varieties:—(a.) *Vegetation;*

(b.) *Fibroid thickening;* (c.) *Atheromatous* and *calcareous degeneration;* (d.) *Aneurism;* (e.) *Laceration;* (f.) *Simple dilatation of orifice;* (g.) *Malformations.*

The exact lesion which causes the morbid state of the valves or orifices must generally, however, remain a matter of conjecture; and it is not of so much practical importance to determine the lesion as it is to determine whether it is of such a nature as to cause obstruction of the flow of blood, or to permit of its regurgitation. One or other of these conditions is indicated by a persistent endocardial murmur, and the persistence of such murmur assuredly points to cardiac hypertrophy and dilatation. Nevertheless, disease at the various orifices operates very differently the one from the other in many particulars, a resumé of which is here given from the admirable treatise of Dr. Fuller.

Aortic obstruction, one of the most common forms of chronic valvular disease, has little effect in producing engorgement of the pulmonary capillaries, or general systemic congestion and dropsy; but when it induces dilatation of the left ventricle, and so causes the *mitral valve* to become so inefficient as to permit of regurgitation, then it is that pulmonary systemic obstruction begins to appear. Aortic obstruction is the least rapidly fatal form of chronic valvular disease. The character of the pulse is not materially altered; but when the action of the heart is forcible, and the obstruction is rough, excessive eddying of the blood may be produced, causing a thrill at the base of the heart, and in the track of the aorta and its branches.

The peculiarity seen in the pulse-tracing from the sphygmograph consists in the obliquity of the line of ascent, which marks the greater duration of the ventricular systole, and the gradual entry of the blood into the vessels. In the following two tracings from Dr. Foster these characters are shown; but as the second was taken from an old woman of over eighty, the modifications of senile change are superadded.

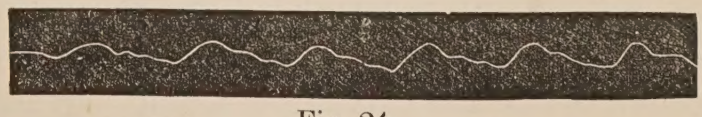

Fig. 24.

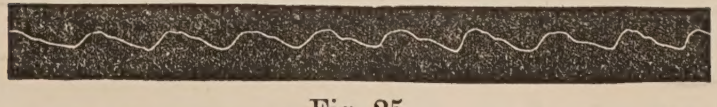

Fig. 25.

Mitral obstruction is comparatively rare. It necessarily induces dilatation of the left auricle. The action of the heart becomes rapid and tumultuous, the lungs become congested, and the right ventricle and pulmonary artery eventually become dilated and hypertrophied. This form of chronic valvular disease induces severe cough, dyspnœa, general distress; and proves rapidly fatal with congestion and œdema of the lungs, and not unfrequently with pulmonary apoplexy. In mitral obstruction the pulse in the earlier and less developed stages is regular and of low tension; is easily depressed and altered in form by pressure on the spring of the sphygmograph; and the line of ascent is oblique rather than vertical.

Obstruction of the pulmonary orifice is a lesion still more rare. It leads to hypertrophy and dilatation of the right ventricle, and ultimately to regurgitation through the tricuspid orifice, with turgescence and pulsation in the large veins of the neck.

Tricuspid orifice obstruction is very rare; and leads to hypertrophy and dilatation of the right auricle, with excessive congestion of the venous system, unaccompanied by any visible pulsation in the neck.

Aortic regurgitation, from incompetency of the sigmoid valves, is one of the most common forms of chronic valvular diseases. It induces hypertrophy of the left ventricle, but does not produce embarrassment of the pulmonary circulation till dilatation of the left ventricle has reached a point at which the *mitral valve* becomes incompetent, and permits regurgitation. It is accompanied by a peculiar and very characteristic pulse-beat. The prolonged swell imparted to the blood at each systole is not sustained. The waves of blood are short and abrupt; the pulse jerks and leaps, and gives a sensation as if successive balls of blood were being shot suddenly under the finger. These peculiarities are consequences of the regurgitation of blood into the ventricle during its diastole. The traces of aortic patency given by the sphygmograph are marked by abnormally great amplitude. The vertical line of ascent marks the sudden ventricular contraction; and is suddenly terminated by a sharp-pointed process (fig. 26, Dr. Foster). The summit of the pulsation-trace is in many cases very short; but in others it presents a horizontal or curved line, especially if any constriction of the aortic orifice exists, or other cause producing delay of the passage of the blood

into the vessels. The second tracing (fig. 27, Dr. Foster) was

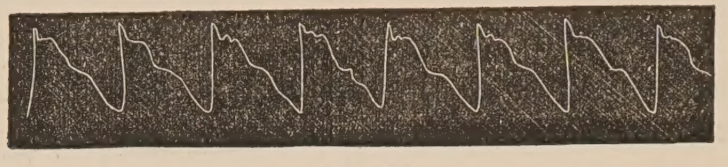

Fig. 26.

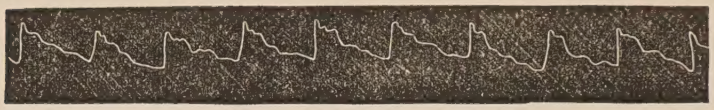

Fig. 27.

taken in a case of aortic patency, complicated with senile change in the vessels.

Dr. Sanderson gives a somewhat different explanation of the same lesion and pulse-tracing (*Brit. Med. Journal*, July 20, 1867, p. 40). "Fig. 28 represents the pulse of a middle-aged man, who had acute rheumatism eight years ago, and several times since. When the tracing was taken, he was suffering from extreme orthopnœa and præcordial pain. The impulse of the heart was to the left of the mammary line, and occupied a space as large as the palm of one's hand. A loud diastolic *bruit* was heard at the fourth cartilage, and a systolic bellows-sound over the aorta. Posteriorly, there was dulness at both bases, and abundant subcrepitant *râles* in inspiration. There could, therefore, be no doubt as to the nature of the case, which soon terminated fatally. After death, it was found that the aortic valve was so altered that the most copious regurgitation of blood must have taken place during each diastolic period. In this case the pulse exhibits characters which Dr. Sanderson believes are not met with excepting in connection with aortic incompetence. "These do not consist, as is often supposed, in the unusual verticality of the expansion; for, as has been already seen, this peculiarity may be produced by merely functional disorder. The distinctive peculiarity consists in the collapse. The tracing shows that the artery becomes completely emptied during the interval between each beat and

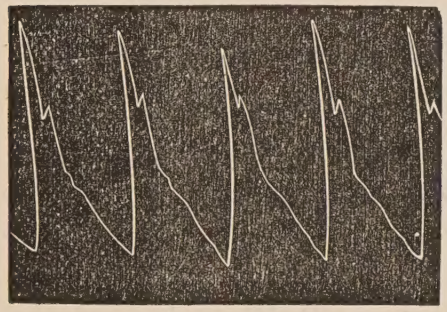

Fig. 28.*

* Pulse of aortic regurgitation.—H. R., aged 36.

its successor; so that the diastolic expansion is no longer indicated. The explanation is simple. Immediately after the heart has ceased to contract, the blood injected into the aorta rushes back into the relaxed ventricle; so that, although the arterial equilibrium is for a moment disturbed, it is almost immediately re-established, the excess of pressure in the great arteries being at once relieved. In other words, the elastic force, which is naturally expended in producing what is called the diastolic expansion, is wasted in regurgitation."

The distinctive features of aortic regurgitation are to be found in the line of descent. There are often one or more extra secondary waves. The first secondary wave occurs earlier than in the normal trace, and the notch which precedes it is often very much exaggerated. But the main characteristic *is the suddenness of the fall of the line of descent, and the comparatively small size of the second secondary wave or true dicrotism* (Dr. Foster).

Mitral regurgitation is the most common of all forms of chronic valvular disease, producing hypertrophy of the left ventricle and dilatation of the left auricle, leading to systolic auricular impulse at the second intercostal space, by admitting of the transmission of the impulse from the ventricle. When excessive, it causes a vibration, a thrill, or a purring tremor, perceptible on the chest-walls in the region of the heart, but which is not transmitted to any extent along the aorta or great vessels. The pulse-tracing is seen to differ from the normal dotted line principally in its great frequency, and in the depth and amplitude of the diastolic notch. This pulse closely resembles the undulating pulse of typhus. In the one disease the contractile force is *weakened*, in the other it is *wasted*. The effect is the same: the systole is ineffectual (Sanderson).

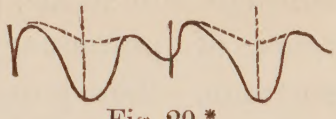

Fig. 29.*

When the regurgitation is also combined with obstruction, from contraction of the auriculo-ventricular orifice, the left auricle usually becomes hypertrophied as well as dilated; and this additional lesion always diminishes the irregularity of the pulse characteristic of pure mitral regurgitation; and the tracing from the sphygmograph is as follows (fig. 30, B. Foster):—

Fig. 31 shows a pulse often seen in rheumatic valvular disease,

* Dicrotic feeble pulse of mitral regurgitation.

with large mitral regurgitation. The heart acts very irregularly. At times the ventricle contracts effectually; but at other times

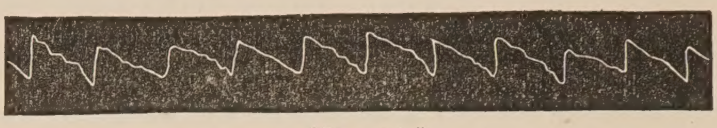

Fig. 30.*

the systolic expansion of the arteries is imperfect and abbreviated, in consequence of which the mean arterial tension declines. Whenever this is the case, the pulse assumes a form which is as

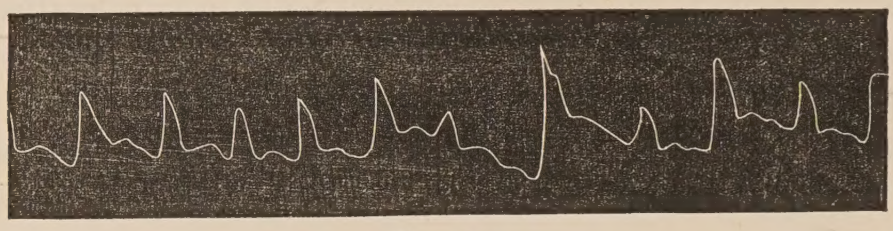

Fig. 31.†

distinctly dicrotic as that of typhus; so much so, indeed, that its double character can be readily recognized by the finger (Dr. Sanderson).

Mitral regurgitation primarily interferes with the circulation through the lungs, producing cough, dyspnœa, and other symptoms of pulmonary congestion; and pulmonary apoplexy is common. The pulse is characteristic. It is irregular in rhythm, and unequal in force and fulness.

Regurgitation through the pulmonary orifice is so rare that its effects have scarcely been verified by clinical observation.

Regurgitation through the tricuspid orifice, although rare as a primary disease, yet is not uncommon as a consequence of dilatation of the right ventricle. The right ventricle then becomes hypertrophied, the right auricle dilated, the *venæ cavæ* distended, and there is a strong tendency to congestion of the systemic and cerebral capillary circulation.

Symptoms.—The symptoms produced by chronic valvular disease mainly depend on the impediment offered to the pulmonic and systemic capillary circulation. In the more advanced stages all forms of valvular mischief result in a certain amount of capil-

* Irregular pulse of pure mitral regurgitation (Foster).

† Pulse of mitral valve regurgitation (Sanderson).

lary engorgement; and therefore all forms of chronic valvular disease have many symptoms in common. Oppression at the chest, breathlessness, speedy exhaustion on exertion, a general sense of lassitude, headache, restless and disturbed sleep, with frequent starting and frightful dreams, cough, palpitation, dropsy, occasional pain in the region of the heart, and sometimes severe *angina*, are amongst the earlier phenomena. The cough is due to pulmonary engorgement, and varies with the amount of *mitral* obstruction or regurgitation. On the other hand, the headache, restlessness, disturbed sleep, and dropsy, are proportioned to the amount of systemic capillary congestion, and vary with the extent of tricuspid obstruction or regurgitation. Albuminuria is not an uncommon occurrence.

The dyspnœa of cardiac disease is peculiar and characteristic, as Dr. Hyde Salter has ably shown:—It is rather a breathlessness than a difficulty of breathing. It has a panting, gasping character. Oppression, rather than tightness, is complained of, and there is a strangling, choking throat feeling about it. The breathing is always accelerated. The dyspnœa is extremely intolerant of movement, or of any exertion whatever, and is often the only circumstance under which dyspnœa is felt. As long as the patient remains at rest there may not be the slightest appearance of dyspnœa, but the moment any exertion is made the breath is gone. The dyspnœa of heart disease is also intolerant of the recumbent posture—hence the name "*orthopnœa*," which signifies "upright breathing"—the patient being compelled to sit erect in order to breathe. In extremely severe cases the patient may not lie down for many days and nights; and should he momentarily fall off into a dose, he is instantly awoke by a sense of impending suffocation, and is in a death-struggle for breath. No suffering can compare with this, and it is not wonderful that the sufferer longs for the sleep of death. "It were indeed a sad story," writes Dr. Ormerod in his admirable Gulstonian lectures, "to tell how patients with disease of the heart die—the tragedies, so to say, of the medical wards of our large hospitals. . . . How some, wrung with pain, have struggled in the week-long agony of death. How some have for days together fixed themselves in the most fantastic postures, the only way in which they could find relief: some leaning forward, resting their hands on a stool, to catch a few minutes' sleep; some on their hands and knees, till the approach

of death, blunting their sensations, allowed them to lie down—a sure sign of coming dissolution."

The dropsy of cardiac disease is usually a late occurrence. It makes its appearance as *œdema* or *anasarca*, very partial and slight at first—a puffiness merely of the eyelids, or more frequently slight œdema of the ankles. By slow degrees it ascends towards the trunk, and ultimately involves the upper extremities and the face; the scrotum in men and the labia in females becoming enormously swollen. Towards the close, effusion is apt to occur into one or both pleura, but ascites is not common (BELLINGHAM). The first appearance of the dropsy, as Dr. Latham observes, marks an eventful period in the progress of cardiac disease. It indicates that a new law takes effect in the circulation, and gains the mastery over the law of health, which has hitherto been able to retain the watery elements of the blood within the blood-vessels. Now, the serum escapes from the blood-vessels, and accumulates in the areolar tissue of the body.

The forms of valvular disease, in the order of the frequency in which dropsy is met with, are,—(1.) Considerable contraction of the left auriculo-ventricular orifice; (2.) Dilatation of the right auriculo-ventricular orifice, with hypertrophy and dilatation of the right ventricle; (3.) A state of the mitral valve and orifice permitting free regurgitation; (4.) Considerable contraction of the aortic orifice. As a general rule, it supervenes earlier the earlier that general venous congestion ensues. A varicose condition of the blood-vessels in the air-vesicles of the lungs is very soon established, which essentially aggravates the dyspnœa.

Prognosis.—It is most unfavourable in cases of mitral and tricuspid regurgitation, and least so in cases of aortic obstruction; and, generally, it may be said that the form of disease which is most rapidly fatal is that which is most rapidly productive of systemic or pulmonic capillary congestion. If the heart be healthy at the date of the occurrence of valvular disease, if the extent of the lesions be not excessive, if the blood be of a normal character, if the viscera be healthy and the secretions free, if the patient's mode of life be regular, temperate, and sedentary, life may be prolonged: but if the heart be hypertrophied and dilated, and if these conditions progress, and if the blood be spanæmic, and the lungs or liver unsound, if the secretions be irregular or defective, or if the patient leads a laborious life, the disease will

run a comparatively rapid course, proving fatal probably within two or three years. In either case death is apt to occur suddenly from syncope.

Treatment is palliative; the object being to regulate and moderate the action of the heart, controlling the tendency to local congestion, and mitigating or removing the symptoms which result from the cardiac derangement. If the patient be plethoric, the heart's action tumultuous, and its impulse forcible, cupping between the shoulders may afford immediate relief to palpitation and oppression at the chest. Repeated or profuse venesection is dangerous, and is calculated to excite irritability of the heart, to impoverish the blood, and to induce dropsy. Repetitions of blood-letting ought therefore to be effected by the application of a few leeches to the *præcordial* region, and very general relief will be obtained by purgatives, aided by dry cupping, mustard poultices, and turpentine fomentations. When, however, active congestion of the lungs exists, venesection, cupping, blisters, and sinapisms are required.

The action of the heart is markedly influenced by the internal administration of *veratrum viride*. The dose of *the tincture* is from *five* to *twenty minims*, which should be gradually increased from the smaller to the greater dose till some obvious effects are produced. If the pulse is reduced, or nausea occurs, no increase of the dose should be made; and if vomiting occur, it should be suspended; and when resumed, the dose should be diminished. *Veratria* is to be given in doses of from *one-sixteenth* of a grain to *half a grain*. When the pulse is sufficiently reduced, the doses should be diminished one-half. *Morphine* or *laudanum*, with brandy, is an antidote for an overdose of this *veratria*, which is an exceedingly powerful remedy as a cardiac sedative, and requires to be used with great caution, the patient being constantly watched. Its depressing effects on the circulation and nervous system are often very striking—a pulse of 75 or 80 being subdued in the course of a few hours to 35 or 40 (*Record of Pharmacy and Therapeutics*, No. v., p. 35, J. C. Braithwaite). *Aconite*, *hyoscyamus*, and *digitalis* are also remedies which exert a sedative influence over the heart.

Sleeplessness being one of the most distressing symptoms, opium in some form might be considered advantageous; but, as Dr. Hyde Salter justly observes, "To give sedatives in

such a case would be the refinement of cruelty. What keeps this poor man awake is not a want of tendency to sleep, but a condition that makes sleep impossible. Relieve him of his orthopnœa and he would be asleep in ten seconds, and so dead asleep that it would take a great deal to rouse him, like a half-asphyxiated child on whom tracheotomy had just been performed. His great struggle, as it is, is the struggle between sleep and life; with opium thrown into the scale of sleep, the struggle for life would only be so much the harder. In one way, and only in one way, would opium give him ease: the narcotic of opium, added to the narcotic of the carbonic acid already circulating in his veins, might accelerate by some hours, or even days, the final coma, and make him sooner sleep the sleep of death. But the euthanasia that is purchased by anticipating the natural process of death comes very near to homicide, and is an alternative that few would adopt" (*Brit. Med. Journal*, Feb. 8, 1862).

Careful regulation of the patient's mode of life is above all things necessary in chronic valvular disease. Excitement of all kinds must be avoided; and the diet should be light, nutritious, and of moderate quantity.

When anasarca supervenes, the hydrogogue cathartics are required to insure copious watery discharges from the bowels. *Gamboge, elaterium, podophyllin,* and *compound jalap powder* should be given on alternate days. Stimulants also may be required, the most suitable being Hollands gin, or whisky.

MYOCARDITIS.

LATIN EQ., *Myocarditis;* FRENCH EQ., *Myocardite;* GERMAN EQ., *Myocarditis;* ITALIAN EQ., *Miocarditide.*

Definition.—*An inflammation of the muscular structure of the heart, extremely rare as an idiopathic disease.*

Pathology.—Cases of inflammation of the muscular substance of the heart from acute rheumatism or pyæmia are not rare; but the cases recorded of other forms of inflammation are too few to enable any satisfactory generalization of their phenomena to be given. Myocarditis is generally a concomitant state, associated with *endocarditis* and with *pericarditis,* and its effects are especially obvious in the strata of fibres nearest the inflamed

membranes. As an idiopathic disease, nothing clinically is known concerning it. Its results may be seen after death in the following lesions :—

(a.) *Suppuration and Abscess.*—M. Simonet has recorded a case of *suppuration* of the heart, in which the disease appeared to result from rheumatism. The patient, a woman, was brought to the hospital labouring under most tumultuous action of the heart, with a pulse irregular and contracted, her breathing oppressed, and her extremities cold. She was bled, but died in a few hours in a fit of syncope. Several purulent collections were found in the substance of the heart, and especially in the interventricular partition. The internal surfaces of the cavities were red in several places: the muscular structure was of a yellowish hue, softened, and easily torn with the least effort.

Dr. Graves was once consulted by a gentleman, fifty-five years of age, complaining of palpitation, dyspnœa, and finally anasarca. He suffered from severe pain and oppression at the region of the heart. Hypertrophy and dilatation of the ventricles were detected. The patient died suddenly a few weeks afterwards; and besides the hypertrophy and dilation, an *abscess* was found in the walls of the heart, which contained about two ounces of pus.

Another case in proof of suppurative inflammation taking place in the heart is one that was examined by the late Mr. Stanley. In this instance the vessels were loaded with venous blood, and the muscular fibres were of a very dark colour, of a very soft and loose texture, and easily torn by the fingers. On a section of the ventricles, numerous collections of dark-coloured pus were seen among the muscular fasciculi. Some of these were seated near to the cavity of the ventricle, while others were more superficial, and had detached the pericardium from the heart. The muscular parietes were softened, and loaded with dark blood.

Suppuration of the muscular substance of the heart and of the coronary artery, in a case of pyæmia, is recorded in the sixth volume of the *Pathological Society's Transactions*, p. 151.

(b.) *Ulceration* of the heart has been occasionally seen, from an abscess in the walls of the heart having opened either into one of its cavities or into that of the pericardium. It has also resulted from the softening of a cancerous tumor, or from a suppurating tubercle. Cloquet has given the case of a man, aged seventy-nine, subject to frequent syncope, who died suddenly, in whose heart

there was an ulceration of the left auricle, through which about two pints of blood had escaped into the pericardium.

(c.) *Rupture and Ramollissement* of the walls of the heart have been occasionally met with. In this affection the heart is flaccid, so that if an incision is made into the ventricles the walls collapse. Its substance tears with great facility. This disease is almost always accompanied by some change in its colour, which is sometimes deeper than natural, and at others, according to Laennec, of a yellowish tint, like that of an autumnal leaf—an appearance which does not necessarily occupy the whole thickness of the muscular substance, but often merely the central layers. This degenerescence is sometimes general, but often partial, affecting only the walls of one ventricle of the interventricular partition, or the walls of one auricle. It is from this cause, perhaps, rather than from any other, that patients sometimes die from rupture of the heart.

Examples of rupture of the right side of the heart are more rare than those of the left; or, according to Bouillaud, there are six ruptures of the left side to four of the right side. Rupture of the auricles is perhaps as frequent as that of the ventricles; or, out of the ten cases mentioned, four were ruptures of the right auricle and two of the left auricle. The extent of the rupture, when it takes place in the ventricle, is various. In one case the ventricle was ruptured from its apex to its base, along the sulcus which separates the two ventricles; in another the rupture was from ten to twelve lines; in a third, the base of the ventricles was severed from the aorta, and one of the aortic valves split transversely. It is remarkable, however, that the rupture has seldom been found at the apex, where the walls of the heart have least force and consistency. The number of the ruptures is as various as their seat; thus, out of forty-eight cases collected by Ollivier, eight were multiple. Again, in two cases related by Rostan, there were two ruptures in each case towards the apex of the left ventricle. Morgagni gives one case, and Portal another, in which there were three ruptures in the left ventricle; and Andral met with a third, in which there were five ruptures; but, of these, three were superficial, only two opening into the cavity of the left ventricle.

Corvisart is the first who has given examples of another kind of rupture of the heart, and it is that of the *carneæ columnæ*, or tendons of the valves. It is probable that rupture of these parts

is owing more frequently to ramollissement, or to induration, than to any other cause. Laennec, however, mentions a case in which it appeared to result from ulceration. In three cases related by Corvisart the rupture followed some violent exertion; and Bertin saw a case in which one of these tendons was ruptured in consequence of a violent fit of coughing. The first symptom in all these cases has been a sudden sense of suffocation, and the patient has in general suddenly died, although in some instances he has survived a few days.

(d.) *Induration* of the walls of the heart is also an occasional lesion of this organ. Bouillaud has collected a series of cases in which this change of structure has been observed. In one, the walls of the heart were almost tendinous. In another, the *carneæ columnæ* of the ventricle were so increased in density as to split. In a third, the walls of the right ventricle seemed to be undergoing a cartilaginous transformation; and Broussais has seen them as hard as a cocoa-nut. Chronic congestion of the heart leads to this condition, and then to defective contractility, leading to dilatation, (Jenner, *Med.-Chir. Trans.*, vol. xliii.) The more usual mode of induration is ossification—a change which usually begins in the coronary arteries, and frequently stops there; but in some rare cases this ossification extends, so that the walls of the auricles, of the ventricles, or of both, and sometimes also of the cardiac partition, become converted into bone. There are specimens in the museum of St. Thomas's Hospital, and many other hospital museums, which make it remarkable how life could have been continued, looking to the unyielding nature and great extent of the ossification of the walls of the heart and great vessels.

HYPERTROPHY.

LATIN EQ., *Hypertrophia;* FRENCH EQ., *Hypertrophie;* GERMAN EQ., *Hypertrophie;* ITALIAN EQ., *Ipertrofia.*

Definition.—*An abnormal increase of the muscular substance of the heart.*

Pathology.—Although occasionally an idiopathic disease, it is more commonly a secondary affection caused by disease of the valves. The hypertrophy may be general or partial—that is, may affect the whole heart or one side of the heart, or one ventricle, or one auricle, or the ventricle of one side and the auricle of the

other, or both ventricles or both auricles, or, indeed, every possible combination of the four cavities. The auricles, however, are much less frequently affected than the ventricles.

The natural thickness of the walls of the left ventricle is in the adult about six and a half lines; but Laennec has seen them, in cases of hypertrophy, to measure an inch and a half, or eighteen lines, in thickness at the base, or triple the healthy standard. This thickness generally diminishes towards the apex, which latter is often natural; but in other cases even the apex is thickened, and instead of two lines it may measure four. *The carneæ columnæ*, and likewise the cardiac partition, are proportionably hypertrophied in these cases.

In hypertrophy of the right ventricle the walls are more uniformly thickened than in hypertrophy of the left ventricle: still, however, the increased thickness is always more marked about the tricuspid valves, and at the origin of the pulmonary artery. The greatest thickness observed has been seldom more than four or five lines, which, taking the natural thickness at two and three-quarter lines, is scarcely a twofold increase. In malformations of the heart, however, it has been found much greater; and both Bertin and Louis have each seen a case in which the foramen ovale was open, and in which the thickness varied from twelve to sixteen lines. Besides an increase of thickness, the walls of the right ventricle, when hypertrophied, acquire a greater firmness, so that on cutting through the walls they do not collapse.

Hypertrophy of the heart seldom takes place without an alteration in the size and form of the chambers. These may, indeed, be natural, but more commonly they are increased; so that, supposing the chamber of the natural size to hold two ounces, when thus diseased it will often contain the larger portion of a pint. This state of parts has been termed *eccentric* hypertrophy; and admitting the normal heart to weigh from eight to eleven ounces, according to age, the weight in hypertrophy is often double or triple that amount; and Bouillaud speaks of eighteen, twenty, and twenty-two ounces being not uncommon. On the contrary, hypertrophy sometimes takes place *concentrically*, or at the expense of the cavity of the heart, and from this cause the ventricle has been found so reduced in size as to be not larger than an unshelled almond.

An analysis of ninety-six cases, collected from various authors, and tabulated by Dr. Sibson (*Med.-Chir. Review*, October, 1854,

pp. 434, 435), shows that by far the larger proportion of cases of valve lesion tend to thicken the walls and enlarge the cavities of the heart; that aortic regurgitation with narrowing of the aperture, and still more without such narrowing, induces active dilatation of the left ventricle, followed consecutively by enlargement of the left auricle and the right ventricle and auricle; that disease of the pulmonic valves causes dilatation of the right cavities; that mitral narrowing with regurgitation leads to enlargement of the left auricle, followed in succession by dilatation of the pulmonary veins, congestion in the lungs, enlargement of the right ventricle and auricle, distension of the venæ cavæ, engorgement of the liver, congestion in the systemic capillaries, and at length, and in nearly one half of the cases, enlargement of the left ventricle itself; that combined disease of the aortic and mitral orifices causes enlargement of the left ventricle, and to a less, but nearly to the same extent, of all the other cavities. Thus there is established a great pathological fact—originally stated by Senac, and confirmed by Morgagni—that *dilatation*, with *hypertrophy*, acknowledge as their cause a force acting *a tergo*, attempting to overcome an obstacle in advance. But there are also other morbid states which, upon the same principle, tend to these results, and are independent of valve disease—namely, *bronchitis*, *emphysema*, and any lung disease in which there is an obstacle to the flow of blood through the lungs. Disease also of the arterial trunks, such as *atheroma*, or morbid states of the blood like anæmia, dilatation or narrowing of vessels, whether of the lungs or of the system, may induce dilatation of the right and left cavities of the heart (ROKITANSKY). The retrogade influence of the systemic capillaries, as shown by Dr. Sibson, tends to exercise a similar influence, as shown by the effect of sudden fright and despair, causing rupture of the left ventricle, or by the influence of Bright's disease altering the quality of the blood, tending thereby to retard its progress through the systemic capillaries. Thus hypertrophy and dilatation ensue from increased resistance to the exit of blood from the cavity of the heart, and from the necessarily increased efforts to expel it, and to propel it onwards. Increased arterial resistance may be connected with a variety of causes. Thus, it may arise either from a contracted state of the capillaries, from diminished elasticity of the arteries, or from narrowing of the aortic valve. Figs. 32 and 33 serve

to illustrate these points. An example of the most simple case of resistance to the contraction of the left ventricle—namely, that which occurs in aortic stenosis—is given in fig. 32. This is the pulse-tracing of a patient in whom the heart, with the adherent pericardium, weighed thirty ounces. The left ventricle was both thickened

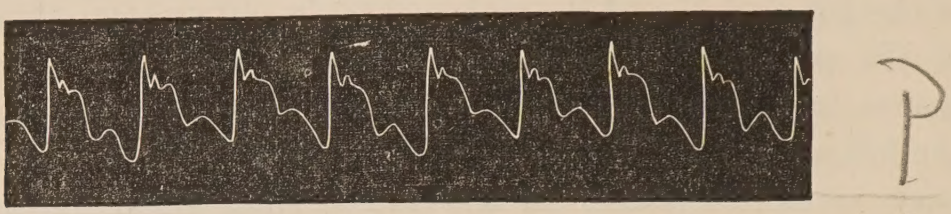

Fig. 32.

and dilated, and the aortic valve so deformed and beset with vegetations, that the orifice would scarcely admit the tip of the index finger. The other valves were healthy. All the other morbid appearances found were distinctly referable to the cardiac lesion as their cause. The tracing scarcely needs explanation. The second event, which, in the pulse of aortic regurgitation, and indeed in all atonic pulses, is either entirely suppressed or difficult to distinguish, is here extremely well marked; and the form of that part of the pulse-curve which represents it shows that the systolic expansion of the artery is of nearly equal duration with the diastolic interval (SANDERSON).

The patient whose pulse is represented in fig. 33 was complaining, at the time when the observation was made, of dyspnœa on the slightest exertion, and præcordial pain. He was subject to nocturnal paroxysms of cardiac distress, which, he stated, always came on with pain at the scrobiculus, followed by palpitation and nausea, with violent respiratory efforts. On examining his chest, Dr. Sanderson found that the cardiac dulness extended from the sternum to the mammary line, and that the præcordial impulse was diffused and expansive. The systolic sound was prolonged, but no abnormal murmur could be made out. The man died several months afterwards, when it was found that the heart weighed twenty ounces, and that the

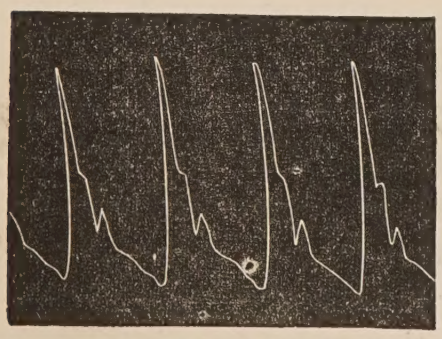

Fig. 33.†

* G. M., aged forty. Pulse of aortic obstruction, with hypertrophy of the left ventricle (SANDERSON).

† W. S., aged fifty-nine. Hypertrophy of the left ventricle without valvular disease.

left ventricle was both hypertrophied and dilated, *without valvular disease.* (See under "Bronchitis" how collapse of the lung may also produce dilatation without valvular disease.)

In detecting enlargement of the heart and thickening of its walls, the size and force of the heart, ascertained by the hand and by auscultation, furnish the principal data. It is by the extent and power of the impulse that the heart's muscular condition is ascertained; and so long as the muscular condition is sound, the valve disease has but little influence on health (SIBSON and STOKES). As a rule, however, the persistence of valve disease implies an enlarged heart, with an impulse increased in extent and in power. But there are also cases to be guarded from mistake—namely, those where a murmur exists with a preternaturally strong, troublesome, quick, and smart impulse, *but limited within a diminished cardiac region.* In such cases such a murmur is of anæmic origin, and the heart is usually lessened rather than enlarged.

Hypertrophy and Dilatation.—Hypertrophy of the heart's substance usually occurs under three forms—namely, (1.) *Simple hypertrophy,* when the cavities retain their relative capacities to each other, although the walls are increased in thickness; (2.) *Eccentric hypertrophy,* when the cavities enlarge or dilate, and the muscular walls thicken—commonly known as *hypertrophy with dilatation;* (3.) When the walls thicken at the expense of the capacity of the cavities—known as *concentric hypertrophy.* The existence of this last form of hypertrophy is now disbelieved, except as a congenital malformation. Cruveilhier, and more recently Dr. Budd, have proved that no such form of hypertrophy exists as a result of disease.

Dilatation of the heart implies that the capacity of its cavities is increased disproportionately to the thickness of their walls. It occurs in three forms—namely, (1.) *When dilatation predominates over hypertrophy;* (2.) *Simple dilatation, where the thickness of the walls are normal;* (3.) *Dilatation where the walls of the heart are attenuated, or thinner than normal.*

Practically, therefore, the physician has principally to deal with the diagnosis and treatment of—(1.) *Simple hypertrophy;* (2.) *Hypertrophy with dilatation of one or more of the cavities;* and (3.) *Simple dilatation with or without attenuation of the walls of the cavities.*

The following table (compiled from Dr. Walshe's treatise) exhibits sufficiently the main points in the symptoms of these forms of disease for the purposes of comparative diagnosis:—

TABLE CONTRASTING THE MAIN SYMPTOMS OF THE FORMS OF HYPERTROPHY AND DILATATION.

A.—General Physical Signs.

Simple Hypertrophy.	Symptoms superadded to constitute Hypertrophy with Dilatation.	General Simple Dilatation.
Arching of the præcordial region, with widening and bulging of the left intercostal spaces from the third to the seventh.		No prominence of cardiac region.
Impulse increased in extent to the left of the sternum.	Extent of visible impulse much greater; pulse may be felt in the back, and its character is less heaving, sharper, and more knocking, than in simple hypertrophy.	Apex beat indistinctly visible or actually invisible, the rounded-off form of the heart destroying the apex form.
Maximum impression felt below and about the left nipple.	Point of the apex beat carried downwards and outwards beyond the line of the nipple, so as to reach the seventh interspace.	Impulse conveys an undulatory sensation to the hand, and feeble in proportion to the purity of the dilatation.
Heart's impulse slow and heaving, as if pressing steadily against an obstacle—in rhythm regular, in force unequal.	Force increased, so as to shake the head or trunk of the patient, or the bed on which he lies.	Force of successive beats is unequal. Rhythm irregular, to a slight or to the very highest degree. The hand applied to the region where the impulse is felt does not feel the beat always strike at the same place.
Superficial and deep-seated dulness augmented in area, but its shape retaining the triangular form.	The dulness tends to assume a square form in place of a triangular one, and may reach from the second interspace to the eighth rib, and from an inch and a half to the right of the sternum to three inches, or even more, outside the vertical line of the nipple. It may be detected in the back.	Intensity of superficial percussion dulness not increased; and in cases of attenuated walls of the heart's cavities, the resistance is less marked than in health. General areas of dulness widened.
First sound is dull, muffled, prolonged, and weakened, almost to extinction, directly over the ventricle.	Sounds gain greatly in loudness and extent of transmission, especially if the valves are not thickened.	*First sound* short, abrupt, and unnaturally clear at the apex and base, appears near the surface, and its maximum point of expression is slightly lowered.
Second sound full and clanging, *post-systolic silence* shortened. Mitral regurgitant murmur, as a clinical fact, exists at one time and disappears at another.	In consequence of the altered direction of the orifice of the aorta to the cavity of the ventricle, a systolic basic murmur may be generated.	*Second sound* not specially affected. Upon the quality of the heart's texture, as regards softness or flabbiness, will depend the extent to which the sounds are transmitted. Intracardiac murmur always regurgitant.

B.—General Functional Symptoms.

Simple Hypertrophy.	Symptoms superadded to constitute Hypertrophy with Dilatation.	General Simple Dilatation.
Strength unimpaired. Power of walking or of ascending a hill diminished, on account of dyspnœa induced by the effort.	Strength tends to become impaired.	Strength fails, and the patient is habitually irritable and melancholy.
Face florid.	Purpleness and lividity great in proportion to the valvular or pulmonary obstruction.	Lividity and mottling of the face prevails, and of the lower extremities generally, with chilliness of surface. Softly pitting anasarca spreads from the feet to the abdomen, external genitals, thorax, face, and neck. Ascites follows anasarca.
Constipation habitual.		Bowels habitually constipated or alternately relaxed—discharges dark.
Dyspnœa occasional.	Paroxysmal attacks of dyspnœa.	Dyspnœa, sometimes called cardiac asthma, becomes complete and habitual, with asthmatic paroxysms, in which the cough is dry, harrassing, and convulsive. Expectoration serous, sometimes streaked with blood.
Radial pulse full, strong, firm, tense, resisting, and prolonged, without jerk or thrill.	Fulness of pulse continues, but strength and power of resistance lost.	Pulse small and feeble, and abnormally late in time after the ventricular systole. It may be regular, or narrow, feeble, fluttering, and irregular.
Pain rare.	Pain not uncommon.	Palpitation and cardiac uneasiness most distressing.
Rarely, and never rapidly, the direct cause of death.	Indirectly, and more or less rapidly, leads to a fatal issue.	Faintness occurs from time to time, and may lapse into syncope and sudden death.

Treatment.—The symptoms of simple hypertrophy may, in the majority of cases, be greatly mitigated by such means as tend to tranquillize the action of the heart. This end may be best accomplished by occasional very moderate cuppings or leeching over the præcordial region. No known drug possesses the power of controlling the growth of the heart. Saline and aloetic purgatives aid the calmative influence of the local abstraction of blood. Diuretics are useful independently of the existence of dropsy; and direct sedatives of the heart's action are indicated throughout, such as *hydrocyanic acid, acetate of lead, digitalis,* and *belladonna;* but of all medicines of this class Dr. Walshe considers *aconite* the best, in the form of the alcoholic extract, given in doses of *one-eighth of a grain.* In repeating the doses, the effects must be watched, while they relieve the painful sensations and disquietude about the heart. If anæmia prevails, animal food should be permitted;

and the more soluble and less astringent preparations of iron should also be given. Fluids must be taken in small quantities; and alcoholic fluids of all kinds are to be entirely avoided. Months and even years of treatment may be required to produce any impression on the disease.

Like *hypertrophy, dilatation* of the heart is not removable by treatment, but judiciously directed remedial measures may render the condition bearable, and even for a time unappreciated by the patient. To improve the tone of the muscle and strengthen the action of the heart, without exciting its irritability, are the objects to be aimed at in the management of the case. The groundwork of medicinal remedies consists in the administration of general tonics in the form of *bitters, mineral acids,* and preparations of *iron.* Belladonna may be employed to tranquillize undue excitement with greater safety than any other sedative remedy. Sedatives, as a rule, are unsafe, and require the utmost caution in their use. Due action from the bowels must be daily obtained, to accomplish which the aloetic medicines are the best, aided by the gentle action of an occasional mercurial aperient.

The diet should be nourishing without being exciting; and may include animal food, with a moderate allowance of light beer or wine in small quantities to dinner.

When dropsy appears, diuretics yield most relief in the form of *acetate, nitrate, iodide,* and *bitartrate of potass, nitric æther, compound tincture of iodine,* the *infusion* and *spirits of juniper,* or *gin,* may all be employed in successive changes, and variously combined. Occasional small doses of blue pill and squill, at bedtime, will facilitate their action generally; and so will cupping over the region of the kidneys, if symptoms of congestion of these organs prevail.

Hydrogogue cathartics also aid the diuretics in subduing the dropsical effusions, in the form of *elaterium, gamboge, bitartrate of potass,* and the *compound jalap powder.*

Dr. Walshe, whose mode of treatment has been here described shortly, prescribes the following formula for the administration of *elaterium:*—

℞. Extract. Elaterii, gr. $\frac{1}{6}$—gr. $\frac{1}{2}$; Extract. Creasotonis, gr. i.; Extract. Hyoscyam., gr. ii.; *misce,* fiat pilula.

(CARDIAC) ATROPHY.

LATIN EQ., *Atrophia;* FRENCH EQ., *Atrophie;* GERMAN EQ., *Atrophie;* ITALIAN EQ., *Atrofia.*

Definition.—*An abnormal wasting and loss of the muscular substance of the heart.*

Pathology.—Care must be taken not to confound this condition with rigid contraction of the substance of the ventricle diminishing its cavity; and, except as a congenital malformation, its idiopathic occurrence is not believed in.

The walls of the heart may be atrophied instead of being hypertrophied, so that this organ has been found to weigh in one case only four ounces two scruples, instead of nine and a half ounces, while the thickness of its parietes was reduced to little more than a thin membrane. This atrophy may be general or partial. In some cases the atrophy takes place without any notable alteration of the capacity of the chambers of the heart. This is termed *simple atrophy of the heart.* More commonly, however, when the walls are thinned, the chambers of the heart are enlarged, and this is termed *eccentric* atrophy. Again, the whole heart may be atrophied and reduced in size, as is often seen in phthisis. Thus, Bouillaud gives the case of a woman, aged sixty-one, whose heart was no bigger than that of a child twelve years old; and Burns gives the case of an adult whose heart did not exceed that of a new-born infant. This form has been termed *concentric* atrophy.

Dilatation of the cavities of the heart may exist both when the substance of the heart is hypertrophied and atrophied; but it may also exist when the walls of the heart are of their natural thickness. In any case the dilatation may be partial or general. Partial dilatation of the heart sometimes presents many curious phenomena: thus, the walls of the right ventricle have been seen divided into two distinct parts; or, as Laennec has described it, into a sort of hour-glass contraction.

In other cases this partial dilatation is perfectly aneurismal. Corvisart gives the case of a young negro who died suffocated, and in whom the superior and lateral part of the left ventricle was surmounted by a tumor almost as big as the heart itself. The inner surface of this tumor contained many concentric layers

of lymph, exactly similar to those of an aneurismal sac. The cavity of this tumor communicated, by means of a small opening, with that of the ventricle. Laennec mentions two cases in which a tumor of a globular form, and the size of a duck's egg, was situated at the point of the left ventricle, and communicated with the ventricle by an opening an inch in diameter. In these cases the left side of the walls of the sac presented a continuation of the muscular fibres of the heart, while on the right side they appeared formed by the two pericardia. Laennec thinks that these aneurismal tumors are formed by ulceration of the internal walls of the ventricle, as in false aneurism of the arteries; others, that it is owing to a separation of the muscular fibres and the protrusion of the inner pericardium.

Hydatids or Echinococcus cysts have been found in the walls of the heart, beneath the inner membrane. Dupuytren found hydatids in the thickness of the right auricle, forming a tumor projecting into the cavity as large as the heart itself. Morgagni found in an old man—who had in no degree suffered from palpitation, syncope, or irregularity of pulse, but had died of acute disease—a cyst the size of a cherry in the walls of the left ventricle. Such parasitic cysts abound in sheep and oxen.

Symptoms of Myocarditis, Hypertrophy, and Atrophy.—Few authors have met with a case of myocarditis, unless complicated with pericarditis, and no distinction has hitherto been observed between the symptoms of these two diseases. Corvisart says it is impossible to distinguish between these affections. M. Laennec affords us no assistance in this dilemma, for he considered that no incontestable example of carditis existed; while Bouillaud says he knows of no symptom which is especially characteristic of carditis (FULLER).

The symptoms of *ramollissement* of the heart are,—a feebler impulse, a slower beat, and greater dulness of the sounds of the heart. Patients suffering from this affection are usually hypochondriacal, liable to palpitation on the least exertion, and often die from the ventricle rupturing.

The symptoms of *induration* of the heart are,—a stronger impulse and a louder sound than usual. This class of patients is greatly subject to *angina pectoris*.

The symptoms of *hypertrophy* of the heart are local and general. The local symptoms are,—a more powerful impulsion, a wider

range of action, and some change in the sounds of the heart. There is also a greater extent of dulness of sound in the cardiac region, and sometimes a bulging out of the left side.

The increased impulsion in hypertrophy of the heart is in proportion to the greater thickening of the walls. Thus, in slight cases, it is only sensible to the hand, while in others the heart "knocks against the ribs," and even raises the head of the auscultator. This greater impulse not only often causes a vibration of the *præcordial* region, but even shakes the whole of the chest. Besides being sensible to the touch, the abnormal action of the heart in these cases is often sensible to sight, each contraction agitating the patient's dress, and sometimes even moving the bedclothes. The point of the heart deviates more to the left, and its motions may be sometimes traced from the second or third rib as low as the sixth or seventh intercostal space.

The increased thickness of the walls of the heart is evidently unfavourable to the transmission of sound; and in simple hypertrophy without enlargement of the cavity, the natural sounds will be duller than in the normal state; and if the hypertrophy be concentric, or with smaller cavities, the natural sounds will be scarcely heard. When, however, the cavities are enlarged, as in eccentric hypertrophy, the sounds are often clear, full, and even much louder than natural.

In hypertrophy of the left ventricle the impulse is stronger immediately under the inferior portion of the sternum than between the fifth and sixth ribs. Lavoisi laid it down as a sign of hypertrophy of the right ventricle that there is swelling of the jugular veins, which pulsate synchronously with the carotids. Corvisart repudiated this symptom; but Laennec found it in every case of hypertrophy of the right ventricle. In general, this pulsation is limited to the inferior parts of the jugular veins, but in other instances it has been seen to extend to the superficial veins of the arm. He regards this symptom, therefore, as one of the best diagnostic signs of hypertrophy of the right ventricle.

In estimating the general symptoms of hypertrophy of the heart, our knowledge of the influence of the left ventricle over the arteries would lead the inference, *a priori*, to softening of the brain; that one of the effects would be a disposition to congestion and to hæmorrhage; and that apoplexy, hæmoptysis, and

hæmorrhage from the bowels would often result. It appears, according to Bouillaud, that out of fifty-four cases of hypertrophy of the heart, eleven, or one-fifth, were attacked by cerebral hæmorrhage, or ramollissement of the central ganglia of the brain. Many suffer from pulmonary hæmorrhage, while a few suffer from hæmorrhage from the bowels. Indeed, on opening bodies that have died of this disease of the heart, the abdominal viscera and mesenteric veins are found loaded with blood. The conjunction of hypertrophied heart is very common in Bright's disease. Besides these concomitants, a pouchy or otherwise diseased state of the aorta often co-exists with hypertrophied heart —the diseased aorta being caused by the abnormal power of the heart; or the hypertrophy of the heart results from a supplemental force being necessary to compensate the functional incapacity of a diseased aorta. Many persons affected with hypertrophy of the heart suffer severely from *angina pectoris*, with palpitation.

The symptoms of *atrophy* of the heart are also local and general. The local symptoms are,—a feeble impulse of the heart, while its sounds are louder, clearer, and more distinct than in health, the intensity of sound being greater in proportion to the atrophied state of the walls, combined with increase of size of the chambers of the heart. The general symptoms are,—slowness of the pulse, occasional palpitation, difficulty of breathing, and tendency to dropsy.

Diagnosis.—The differential diagnostic symptoms of myocarditis, of ramollissement, and of induration of the heart, are imperfectly known. On the contrary, the symptoms of hypertrophy of the heart, and of enlargement of its chambers, are so striking that it is impossible to mistake them; but it should be remembered that they are often latent, unless aroused by some mental emotion or sharp exercise—a fact necessary to be remembered in the examination of recruits, or of lives for insurance offices.

Prognosis.—It is probable that diffuse inflammation of the substance of the heart may exist, and may be recovered from; but if any morbid product forms in its substance, the prognosis is eventually an unfavourable one. Ramollissement, as well as induration of the heart, from the tendency to rupture in the one case and to ossification in the other, if they can be determined to exist, are always of grave prognosis, although the patient,

perhaps, may survive many years. Hypertrophy, atrophy, or dilatation of the heart, are perhaps compatible with health till dropsy or hæmorrhage takes place, when the conjoint diseases are either most difficult of cure or fatal.

DEGENERATION OF THE MINUTE TISSUE OF THE HEART.

Definition.—*A change in the muscular substance of the heart, which results in the elements of the muscular fibres being replaced by molecular particles, chiefly fatty or fibrinous (fatty or fibroid degeneration). The change tends to sudden death by rupture of the heart, or by syncope.*

Pathology and Symptoms of Cardiac Degeneration.—Microscopic observation has revealed certain remarkable changes, of the nature of degeneration (see vol. i., p. 112), in the heart, as well as in other organs, of slow and insidious development, most difficult to detect during life, hitherto unassailed by any remedy, productive of most fatal consequences, and the immediate cause of the sudden and unexpected demise of many distinguished men. It has especially cut off the hard-working men of the intellectual class—*e. g.*, the Rev. Dr. Chalmers, and Dr. Abercrombie, of Edinburgh; Dr. Pereira, of London; and very recently Sir Cresswell Cresswell and Sir Frederick Slade. Improved means of diagnosis have taught us to anticipate such a termination to many cardiac affections with which we are every day familiar; and in convalescence from severe injury, or under chloroform, sudden death is extremely apt to supervene by syncope, in cases where the heart has undergone the degenerations about to be described.

At least two varieties of fatty disease of the heart have been recognized. In the one form the fat, composed of oil in nucleated cells—the ordinary fat-cells—grows on the surface of the organ, between its muscular fasciculi and the reflected pericardium, especially at the junction of the auricles and ventricles, along the trunk of the coronary veins, at the edges of the ventricles, at the apex, and at the origin of the aorta and the pulmonary artery. The right ventricle is often almost entirely covered with fat. This form of fat accumulation is not degeneration; but it so gradually encroaches on and insinuates itself between the muscular fibres, that it conceals, impoverishes, and ultimately

causes them to waste, so that the muscular walls become thin, especially towards the apex and over the walls of the right ventricle. In these parts the fibrous structure almost disappears, and the *columnæ carneæ* appear to spring altogether from the *endo-pericardium.* In this form of fatty heart the muscular fibres may remain healthy, although they sometimes eventually degenerate.

In the other form of fatty heart a degeneration of the fibre ensues. Its muscular element disappears, and its place is taken by fat in a molecular form, and minute oil-globules ultimately come to fill the sheaths which previously contained muscular fibre. But it is now observed that various forms of degeneration are capable of microscopic demonstration in the minute fibrillæ of the heart's substance, the result of decay or disintegration.

The recognition of fatty degeneration in the minute tissue of voluntary muscle led to the observation of analogous changes in the tissue of the heart by Corvisart and Laennec. In Scotland the younger Duncan, Cheyne, and Adams were amongst the earliest observers; but the subject has been mainly elucidated by the writings of Smith, Stokes, Andral, Rokitansky, Hasse, Paget, Ormerod, Quain, Begbie the elder, and Handfield Jones. Considerable variety of opinion prevails amongst these observers regarding the nature and the sequence of phenomena associated with this degeneration: and especially as to the symptoms diagnostic of fatty degeneration of the heart, by which its existence may be inferred during life.

I have made an analysis of twenty-nine cases of sudden death associated with such a lesion, the histories of which are scattered throughout the first ten years' "Records of the Transactions of the Pathological Society of London" (*Med.-Chir. Review*, 1858, p. 429). The results of this analysis are incorporated in the following account, illustrative of the pathology of this formidable disease:—

Age.—Of the twenty-nine cases sixteen were females and thirteen were males, in all ranks and social conditions of life. The average age of the females was *forty-six* years, and the average age of the males was *fifty-two.* The youngest patient was a male infant six months old; while the oldest male and the oldest female appear each to have been *seventy-six* years of age. The youngest female was *ten* years of age; and the youngest male (exclusive of the infant) was a boy eleven years of age.

In the female a tendency to the degeneration seems to have been observed at a much earlier age than in the male; but between the ages of *fifty* and *eighty*, in both sexes, the greatest number of cases have occurred. The Rev. Dr. Chalmers's age was *sixty-eight;* that of Dr. Abercrombie was *sixty-five;* that of Sir Cresswell Cresswell was *seventy.* Among eighty-three cases collected by Dr. Quain, death occurred in fourteen between the ages of *fifty* and *sixty;* in eighteen between *sixty* and *seventy;* and in fourteen it occurred between *seventy* and *eighty.*

The general health and condition of the patients previous to their fatal illness has been variously described. In eight of the cases recorded in the Society's *Transactions* the previous health is described as delicate, weak, nervous, or reduced by previous illness, such as miscarriage, menorrhagia, hæmorrhoids. The patients who are thus described were of short stature, thin, and spare make. Eight other cases are described as strong, stout, fat, muscular, or hearty. Of these some were of sedentary habits, unaccustomed to active employment, but temperate in habit. Others had suffered from slight attacks, such as of bronchitis, "liver complaints," "spasmodic pains of the stomach." Some are described as anæmic and pale, although at the same time stout and well-grown.

A third class of cases had suffered more or less severely from previous attacks of acute diseases, although they had recovered from them to some extent. One had suffered from occasional headache, with seizures of an apoplectic-like nature after fifty-five years of age. A similar case is related by Dr. Fuller (*Diseases of the Chest,* p. 602). Another had been thirty years in India, exposed to malaria, and had suffered from frequent attacks of intermittent fever. A boy, aged ten, had fever at the age of four years, afterwards chorea, and subsequently scarlatina and rheumatism five months before death. Mental anxiety and domestic distress, inducing great irritability and nervousness, were the antecedents of two other fatal cases; and five other cases are described as having to all appearance enjoyed excellent health.

Dr. Begbie, senior, in a most interesting communication to the Medico-Chirurgical Society of Edinburgh (January 15, 1851), relates some passages in the lives and deaths of the Rev. Dr. Chalmers, and of Dr. Abercrombie, which bear on the previous general health of these two distinguished men. Of the Rev.

Dr. Chalmers he relates, that "to a mind of the highest order, and of wondrous energy, he united a hale and vigorous, a manly and robust frame. He spared no exertion, either mental or physical, in carrying out the great object of his life. He was hardly ever incapacitated by infirmity or loss of health in prosecuting his enterprise; and from early manhood to green old age, even up to his latest hour, he toiled and spent his energies and strength."

These things Dr. Begbie mentions, "to show that the fatal disease which lurked within, which was progressive in its nature, and probably of long standing, could neither have produced serious uneasiness nor proclaimed its presence by any unequivocal signs." But it is related that, thirteen years before his death, Dr. Chalmers had a sudden seizure on the street, of what proved to be a serious and alarming illness. He lost the power of the arm and leg of the right side, and experienced diminished sensation on that side. His face was pale, the skin cool, the pulse soft and frequent. After a few weeks of rest and quiet he completely recovered, and returned as vigorously as ever to his professional duties; but with accumulating years there came a disposition to obesity; and with the silver grey on the massive forehead came also the pallid and somewhat sickly look of fading health. He was sometimes sick at stomach from some trival ailment arising from indigestion; but he was never faint, nor ever swooned away.

Of Dr. Abercrombie it is related that he enjoyed, during a long series of years, uninterrupted health; but three years before his death he was suddenly seized with loss of power and impaired sensation over the left side, but without the loss of consciousness or any affection of speech. He experienced great anxiety, and complained of præcordial uneasiness and slight headache. He sighed frequently, and had a cold skin and pallid countenance. The pulse was frequent and small at first; but after a while it subsided in frequency and rose in strength. Dr. Abercrombie's own impression was that his illness was paralysis, connected with cerebral disease. It never occurred to himself or his medical advisers to connect the symptoms with deranged circulation arising from a damaged heart; and although he complained of præcordial uneasiness, which never amounted to actual pain, and of something more than uneasiness in the left arm and shoulder, and at the base of the scapula, yet the

circulation was regular, though feeble—a feebleness which might have been accounted for by the active depleting measures he himself employed, and the scanty fare to which he subjected himself. Although he recovered from this attack he continued pallid; and just before his death he was observed to be breathless on ascending a stair.

In the cases detailed in the *Transactions of the Pathological Society*, *cardiac symptoms* occurred sometimes suddenly after exposure to cold, with pain in the chest, shortness of breath, and palpitation. In some of the cases associated with *angina pectoris* the pain was sometimes excessive, shooting down the left side and arm, especially after any excitement. In other cases there were *cardiac symptoms*, consisting of a dull pain in the region of the heart or ensiform cartilage before death, associated with dyspnœa. In some *angina pectoris* prevailed for many years. The cardiac affection in several instances betrayed itself by cough, dyspnœa, and general debility, a sense of oppression at the chest, and desire to draw a deep breath, the breathing being sometimes embarrassed and aggravated by exertion. In seventeen out of twenty-nine cases death was sudden and unexpected, and cardiac disease had never been observed, and only in one case suspected to be present, by the medical attendant.

In most instances the attacks of giddiness and apparent coma are due to syncope, causing a deficiency in the supply of blood to the brain, consequent on feebleness of the heart's action. Unlike what happens in cases of apoplexy connected with hypertrophy of the left ventricle and excitement of the arterial circulation, the patient when attacked is pale, has a feeble pulse, and presents more or less lividity of the lips—symptoms which may serve as indications of the true nature of the disorder (FULLER).

The condition of the pulse having been observed after the cessation of an acute attack of disease, in twelve cases out of twenty-nine, which afterwards terminated fatally, it was noted as "irregular and unequal;" as "feeble, rapid, and irregular;" as "feeble and intermitting, or occasionally so every eight or ten beats;" as "large, jerking, and compressible, one hundred and six per minute—afterwards a hundred, but small, jerking, and regular to the last;" as "small and feeble, but no irregularity or intermission." In one of the cases the pulse was observed for

two years and a half previous to death to be irregular and uncertain in its action—a condition which disappeared during an attack of bronchitis, but which reappeared as the patient recovered from the bronchitic attack—varying from *seventy* to *eighty* in a minute. In other cases, where its condition had been long noted, it is stated to have been weak, irregular, and intermitting, numbering at first *eighty*, becoming weaker, often intermitting, and more slow (*sixty* to *seventy*) shortly before death, or small and feeble. The slowest pulse recorded is *fifty-five*. In one case of death under the influence of chloroform, the pulse at first was *ninety-four*, regular, and of average firmness: subsequently, under its influence it was accelerated, and suddenly began to get smaller, weaker, and imperceptible. In extreme cases the pulse may fall as low as twenty-six or thirty in a minute, owing to the failure of certain systoles of the heart to communicate a pulsation to the radial artery, the heart itself beating at the rate of fifty-six or sixty in a minute (FULLER, C. J. B. WILLIAMS, GAIRDNER).

The sounds of the heart have been observed to be modified. In one case "a loud bellows murmur" was heard all over the chest; death followed after complete destruction of the mitral valve by rupture. In another case there was extensive dulness over the heart, and a loud bellows murmur with the first sound; and in this case the edges of the mitral valve were fringed with bead-like vegetations. In a third case a *diastolic bruit* existed in the region of the aortic valves, the heart's action ultimately becoming tumultuous, and the sounds obscured, attended with a rolling action four days before death. In this case a band of lymph was found extending across the aortic orifice, with irregular vegetation over the sigmoid valves, while at the opening of the coronary artery a false aneurism opened into the muscular substance of the left ventricle. In a fourth case the systole was attended with a loud and prolonged *bruit*, loudest over the mitral valve, followed by a distinct natural second sound, to which succeeded a remarkably prolonged interval of rest, and the rhythm was frequently irregular. Associated with these sounds was some thickening of the aortic valves, and especially a peculiar degeneration into fibrinous matter of the muscular substance towards the base of the heart. In a fifth case the area of the *præcordial* dulness was *less* than natural, and there was a systolic endocardial

grating murmur, with feeble impulse, and the mitral valve thickened.

In four only out of the twenty-nine cases were the cardiac sounds so modified that (in the absence of valvular disease) the cause of the modification was ascribed to the morbid condition of the muscular tissue of the heart. In one of these cases the impulse and sounds of the heart were observed to have been feeble six weeks before death. In a second case the impulse was noted to have been feeble and of limited extent, the second sound being very indistinct, but no murmur. In a third case the heart's action was feeble and irregular, the sounds being weak; and in a fourth case the sounds were muffled, but no *bruit* existed.

Other symptoms associated with this remarkable degenerescence and formidable disease were,—in some cases severe vomitings and faintings, or a peculiar sinking and sense of faintness, with profuse perspirations; paleness and lividity, with urgent dyspnœa for some weeks before the fatal result; sudden aggravation of cardiac symptoms, of cough, or of general debility, for some days before death. In one case there was evidence of scurvy or of purpura; and thirteen days before death this patient became unsteady in gait, had impaired vision, with a vacant countenance, dilated and sluggish pupils, headache, and pains round the orbits, with spectral illusions, partial blindness, and hæmorrhage from the nose and bowels (Bristowe, *Path. Society's Trans.*, vol. v., p. 93). In another case the general symptoms consisted of slight dyspnœa, with evidence of acute rheumatism; and four days before death dyspnœa became urgent. In a fourth case, for four years previous to death, sudden and severe attacks of shortness of breath were observed on exertion. These attacks were relieved by *ether* and *ammonia*, but they were always followed by faintness and exhaustion, with coldness and lividity of the face and extremities (Quain, *Path. Society's Trans.*, vol. iii., p. 82). In the fifth case, what is termed "biliousness" was troublesome, with a sense of heaviness and oppression referred to the sternum. Two months before death there was general *malaise*, numbness of the fingers of the left hand, with tingling or uneasy sensations over the surface generally. A sixth case suffered from attacks of headache at intervals of a few months, associated with flashes of light before the eyes, and darkness of half the objects seen. Such attacks lasted about a week, attended with loss of speech, impairment of

memory, and feebleness. In three cases, attacks of *angina pectoris* are recorded. In one of these, six weeks before death, the attacks became so violent that the patient lived in constant dread of pain, which was of a most excruciating kind, accompanied by a feeling of suffocation. Three days before death he attempted suicide by cutting his throat in one of these paroxysms; but the wound was very superficial, and healed. A convulsive fit continued for ten minutes two hours before death.

The modes of death may be described as follows:—

In *eleven* out of twenty-nine cases the death was absolutely *sudden* or *instantaneous: two* were *comparatively sudden in bed,* the patients having been previously up at the night chair; *three* patients were found lying dead; and in *three* a death-struggle existed of from *three* to *five* minutes' duration; in *one case* four paroxysms of rigor, nausea, and "spasm of the stomach," with small and contracted pulse, occurred at intervals within twenty-four hours, and at last suddenly proved fatal; another patient died in a paroxysm of *angina pectoris;* another under the influence of chloroform.

Of the cases of absolutely sudden or instantaneous death, *nine* died from rupture or laceration of some part of the texture of the heart. Such was the mode of death of Dr. Abercrombie: a rent half an inch in length existed in the posterior aspect of the left ventricle, from which the pericardium was suddenly filled with blood. Such cases die of broken hearts—literally, not figuratively. Their cords are either torn asunder, or the fibres and minute vessels of the substance of the heart are lacerated. In one instance there was sudden rupture of the mitral valve. In five, the substance of the left ventricle was ruptured, two at the upper and posterior part, three across or along the anterior wall, and more or less close to the septum. In one there was rupture of the septum itself, penetrating the right ventricle.

Death by syncope is another usual mode of death in such cases. Thus died Dr. Chalmers, the eminent Scotch divine. It is a mode of death apt to occur in cases of convalescence from injury; and the cases of Dr. Pereira, who died while convalescing from rupture of the *tendo Achilles,* and of Sir Cresswell Cresswell, who died while recovering from an injured patella, might suggest the necessity of a cardiac examination in similar cases, and the maintenance of a sufficiently stimulating diet, and wine, if previously

taken, with a careful avoidance of such circumstances as may tend to syncope, in handling the injured parts of men otherwise healthy, but advanced in years.

Morbid Anatomy and Nature of the Change observed in the Minute Tissue of the Heart.—A considerable variety of description under this head has been given, which may be classified as follows:—

1. *Cases in which there is an interstitial deposit*, exudation, or growth of material, thickening or indurating the substance of the heart, and in which the new material and the sarcal particles within the muscular sarcolemma alike undergo degeneration (Risdon Bennett, Bird, Ogle, *Path. Society's Trans.*, vol. iii., pp. 273, 276, 281). In such cases there was evidence of previous or of existing pericarditis, and sometimes of rheumatism; the heart was hypertrophic, increased in weight, and the fibrillæ exhibited undoubted evidence of extreme brittleness (Barlow, *Path. Society's Trans.*, vol. iv., p. 71; Bristowe, Peacock, Cholmeley, l. c., vol. v., pp. 84 and 102; vol. vi., pp. 147 and 148). A section showed the muscular substance of the walls of the ventricle to be increased in thickness, encroached upon, and in part replaced by some adventitious product. Towards the base, the muscular substance gradually disappeared at the expense of its outer part, so that at the distance of three-quarters of an inch from the aortic valves, and from that point upwards, it was entirely wanting, and was replaced by a firm, dense, slightly translucent fibrous tissue, which extended some little way upwards on to the aorta, and downwards on the exterior of the muscle, gradually losing itself in the substance of the pericardium.

Several masses of deposit existed in the muscular substance, of an irregular form, opaque, yellowish, and somewhat firm. This deposit consisted of cell-elements, fatty particles, and degenerate muscular fibres. The cells were spherical, and varied in size from $\frac{1}{1000}$th to $\frac{1}{200}$th of an inch in diameter, having very thin walls, easily broken down, and containing a few granules. Some larger cells showed nuclei in their interior; and they were arranged in lines, so as to give the appearance of fibres. This deposit was considered of a doubtful nature as to its being malignant, tuberculous, concrete pus, or fibrinous deposit, such as is sometimes seen in the spleen. This latter view was adopted by Dr. R. Bennett, Mr. Simon, and Dr. Quain. The deposit in the heart resembled a similar lesion figured by the late Sir Robert Carswell

in the fourth fasciculus of his "Morbid Anatomy," plate 3 (*Path. Society's Trans.*, vol. iii., p. 273). In some cases the deposit or altered part appears to be circumscribed by a boundary line, where blood-globules are abundantly present; and had the process of softening continued with surrounding vascular activity, "a circumscribed abscess must have been the result; if absorption had taken place, a fibrous degeneration would have been left" (QUAIN, l. c., p. 281). The simultaneous occurrence and progress of both these forms of morbid process has also been observed (OGLE, l. c., p. 282). In one case, where the heart weighed *twenty* ounces, the lesion appeared to consist of lymph amongst the muscular tissue, which lymph becoming converted into more condensed fibroid material, compressed, and ultimately replaced, the muscular substance (BRISTOWE, l. c., vol. vi., p. 150).

Dr. Henry Kennedy, of Dublin, has observed that *enlarged heart* forms an important element in the natural history of the affection (RANKING, *Abstract*, vol. xxx., p. 93).

2. *Cases in which there is a very moderate amount of degeneration of tissue, or alteration in the bulk of the heart*, but in which the functional disturbance is serious in the extreme, and the case rapidly fatal in its issue (WILLIAMS, l. c., vol. ii., p. 186).

3. *Cases in which, amongst apparently healthy tissue, a portion of the muscular substance has undergone degeneration*—the evidence of degeneration consisting of "the disappearance of the cross-marking of the muscular fibrillæ," and "the fibres being filled with oleo-albuminous or fatty granules." The coronary artery in such cases is found ossified or obstructed, going to the seat of degeneration (QUAIN, l. c., vol. ii., p. 188; and vol. iii., pp. 262, 270, 273). In the case of the Rev. Dr. Chalmers, Dr. J. H. Bennett, Professor of Physiology, of Edinburgh University, made the *post-mortem* examination, and reported that the substance of the heart throughout consisted of fatty granules. The muscular fasciculi could scarcely be seen, although here and there traces of the longitudinal fibrillæ could be observed. No transverse striæ were anywhere visible. The heart was flabby and unusually soft. The coronary artery was loaded with calcareous deposit, much contracted, and in one place obliterated, presenting considerable resistance to the knife.

4. *Cases in which the death could not be ascribed to fatty degeneration of the heart alone, but where other lesions existed*—

such, for instance, as fatty degeneration of the cerebral arteries, resulting in death by apoplexy; or in cases in which apoplectic seizures occur—a lesion we owe so much to Mr. Paget for elucidating; or in which, according to Dr. Quain, obstruction to the flow of blood from the head leads to congestion of the brain and hæmorrhages. The source of the obstruction Dr. Quain believes to be due to the want of power of the right ventricle (on account of degeneration) to maintain the circulation through the lungs; the blood, therefore, accumulating in the brain, tends to death by apoplexy, for the most part meningeal (l. c., p. 190).

5. *Cases in which, in addition to the degeneration of the heart's fibres, they are also encroached upon and rendered powerless by the growth of fat intruding upon them, and covering them up.*

Atrophy, wasting, and disappearance of the proper muscular tissue are the immediate results of this encroachment, terminating in a thinning of the muscular parietes of the organ (l. c., vol. i., p. 192). Along with this especially atrophic result, some of the fibres of the heart which remain undergo the fatty degeneration within the sarcolemma; but this homogeneous sheath seems to remain intact, while the nuclei within or upon it disappear, or break up into streaks of oil dots. Thus, true "sarcous elements" ultimately come to be replaced by minute opaque molecules (the nature of which has not been in all cases determined), or actually by small drops of oil.

6. *There are cases of this degeneration which result in the "cardial apoplexy" described by Cruveilhier, where hæmorrhagic spots, or extravasations of various sizes, occur in the substance of the muscular tissue; the surrounding tissue is found in the state of fatty degeneration, and the coronary arteries leading to the degenerate tissue and hæmorrhagic spot are ossified or obstructed* (QUAIN, l. c., vol. i., p. 192; vol. ii., p. 190). A condition somewhat of this kind seems to have occurred in the case of Dr. Abercrombie. The late Mr. John Goodsir (Professor of Anatomy in Edinburgh University), who made the *post-mortem* examination, reported that irregular ecchymosed spots were situated near the rupture which proved fatal. These spots consisted of effused blood; but their connection with ruptured vessels could not be distinctly made out. The serous membrane over them was quite entire. Both coronary arteries were much dilated as they passed from the aorta; and throughout their course they contained in

their walls much atheromatous matter. The heart was slightly enlarged and dilated, rather loaded with fat, and remarkably soft, as if from interrupted nutrition. Mr. Goodsir examined the tissue near the rupture with the microscope, and found that the muscular fibres had undergone the fatty degeneration. The granules were arranged in transverse rows, and some of the fibres were nearly empty. To the naked eye the muscular tissue of such hearts presents a pale, mottled, or dirty yellow appearance. It has none of the flesh-red hue of health. The heart is soft and flabby to the touch; and its texture at the degenerate parts so friable, and sometimes brittle, that it yields to the slightest pressure, and may be torn without difficulty.

The elasticity of the muscular substance is so completely lost that, when cut across, the walls at once collapse.

Diagnosis.—The diagnosis of a degenerate heart is by no means easy; and a diagnostic value having been attached to the existence of fatty atrophy of the cornea—the "*arcus senilis*" —in connection with this disease, it is necessary to notice the circumstance only to mention that, as a sign of fatty heart, it is by no means to be depended upon in every case. (Watson)

Treatment.—Iron in its various forms, quinine, and mineral acids are the medicinal agents suggested by the nature of this disease. Freedom from anxiety, thorough repose of mind, entire avoidance of fatigue, gentle and regular exercise in the open air, careful attention to the state of the skin, a generous and stimulating diet at regular intervals, in moderate and equable amount at each meal, are the main hygienic indications calculated to impart tone to the system, improve the condition of the blood, and so induce a more healthy nutrition of the heart (FULLER). A salt-water sponging bath should be used daily. Most druggists supply the saline materials for such baths. In cases where digestion is sufficient, cod-liver oil, cream, and milk may be given with great advantage (TANNER). The bowels should be so regulated as to render straining at stool unnecessary.

Living as we do in a tumult of incessant excitement, hurry, and competition, "the struggle for existence" is maintained till the heart fails almost unperceived. The Physician can but indicate the fact; and in the life that we now live, the disease, if it is not more frequent, is certainly obtruding itself more on public notice than hitherto.

Watson gives a capital acct of the symptoms with cases

ANGINA PECTORIS.

LATIN EQ., *Angina pectoris;* FRENCH EQ., *Angine de poitrine;* GERMAN EQ., *Angina pectoris;* ITALIAN EQ., *Angina pectoris.*

Definition.—*Pain or spasm of a weakened heart* (CHEVERS), *referred to the lower part of the sternum, or to the præcordial region, extending through the chest to the left scapula, and up the sternum to the root of the neck. The pain is characterized by its suddenness, its severity, and by a sense of constriction or of burning. It compels the patient, if walking, instantly to stop,—almost prevents inspiration. The pain is felt likewise in the left shoulder, whence it sometimes reaches to the elbow, rarely to the hand, often with a sensation of numbness in the parts. A tendency to syncope exists, associated with intense anxiety, and a sensation of approaching dissolution.*

Pathology.—This disease had attracted little attention till Dr. Heberden, in 1772, drew the attention of the profession to it by two papers published in the second and third volumes of the *Transactions of the London College of Physicians.* He connected it with disease of the heart; and it has ever since been treated of in conjunction with diseases of this organ. It has subsequently been studied by Drs. Black, Parry, and Jenner, and by many Continental physicians; and Dr. Parry's work—*An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris*—will well repay perusal, though published so long ago as 1799.

The immediate cause of the paroxysm, as shown by the most weighty testimony, appears to be a sudden impediment to the *coronary* circulation of the heart, and particularly to the return of the blood by the coronary veins. These results are in general due to a temporarily over-distended state of the chambers of the heart, and an inability in them to empty themselves, whether owing to weakness of the muscular parietes of the left ventricle or to other causes. If the cavity of the left ventricle is considerably dilated, or its walls attenuated or softened, or otherwise degenerate, the contractile power of its muscular tissue will be impaired in proportion. If the circulation under these circumstances happen to be suddenly hurried, or the heart's action suddenly disturbed, the cavity of the left ventricle may become

so much distended as to render it incapable of contracting efficiently upon its contents, which would be quickly followed by distension of the auricle on that side, and, if relief were not soon experienced, by distension of the right side of the heart (BELLINGHAM).

The organic lesions of the heart most apt to be attended by *angina* are conditions of the aortic valves which permit of free regurgitation, with a rigid, dilated state of the ascending portion of the arch of the aorta, which permits the blood from the large vessels to regurgitate into it, combined with either of the following conditions of the left ventricle:—(1.) Dilatation of the cavity; (2.) Attenuation of the parietes; (3.) Softening or degeneration of the muscular tissue (BELLINGHAM).

Any one of those morbid conditions may be present, or two or more of them may be combined, and yet *angina* may not necessarily occur, so long as the circulation continues tranquil, and so long as the left ventricle is able to get rid of the blood which enters its cavity, and does not get over-distended. But if the heart's action becomes disturbed by some sudden mental emotion or other cause; or even without this occurring, if the stomach is loaded with indigestible food, and it and the intestines are distended with flatus, by which the cavity of the chest is encroached upon, and the heart's movements are impeded, a paroxysm of *angina* is the general result. John Hunter, who suffered greatly from this disease, used to affirm that his life was in the hands of any person or circumstance which acted powerfully on his mind; and, in fact, he ultimately died in St. George's Hospital, from strong but suppressed feelings on a point in which he was interested. Ascending a staircase or other acclivity, or indeed any active exertion, is a powerful exciting cause. In persons who have had previous attacks, the paroxysm is liable to supervene during sleep, as the result of a frightful dream disturbing the heart's action, or of considerable distension of the stomach by flatus impeding the movements of the heart.

The late Sir John Forbes, in an able article on this subject in the *Cyclopædia of Practical Medicine*, has shown that *plethora* becomes a very common complication of *angina pectoris*. The very existence of *angina* tends to produce *plethora* if it did not previously exist; a sedentary life and abandonment of all active bodily exertions are almost inevitable consequences of the disease.

Angina pectoris ought therefore to be regarded rather as a *symptom of organic disease of the heart* than as a distinct form of disease. What *dyspnœa* is to the lungs, *angina* appears to be to the heart; so that Bellingham has termed it the "dyspnœa of the heart."

Symptoms.—The paroxysms of this disease generally supervene suddenly, and are characterized by a constrictive anxious pain, fixed most commonly on the left lower half of the sternum, and rarely extending above the fourth rib. Occasionally, however, it extends over the whole anterior portions of the chest, along the neck to the lower jaw, into the back and shoulder, down the arm to the elbow, and even to the hand and fingers. In this course it affects externally the superficial cervical plexus and its ramifications, as well as the anterior thoracic nerves, the cubital nerve, and its divisions. The pain is also sometimes sub-sternal, and then follows the course of the nervous plexus placed between the folds of the mediastinum, and also the branches of the eighth pair which go to the large arteries and surround the bronchial tubes. The pulse is sometimes rapid, sometimes hardly to be felt; and the breathing is sometimes accelerated, at other times it is imperceptible. Mr. Hunter, when labouring under the paroxysm, could scarcely feel his pulse, and thought he should die unless he exerted his voluntary muscles to aid respiration. Many have died so—asphyxiated. Darwin has seen the action of the diaphragm, and consequently the phrenic nerves, affected; while Laennec mentions that the lumbar and sacral nerves also partake of the pain. Besides the parts which have been mentioned, the gastric system is also much affected, the patient perhaps being in an instant distended with wind, and only relieved by repeated eructations. The urine is sometimes suppressed during the paroxysm. In all cases where the patient is not broken down by disease, the mind is clear, but the face and extremities are cold and pale. At length the paroxysm subsides gradually, when much wind is discharged, accompanied by a copious and almost involuntary flow of pale, limpid urine, and the patient for the time recovers.

The duration of the paroxysm depends on the persistence of the impediment to the coronary circulation. Sometimes the pain only lasts a few minutes, while at other times it will continue for two or three hours, a whole day, or even longer.

The interval of respite from pain is likewise very uncertain —from a few hours to a few days, or a few months. Each repetition, however, increases the tendency of the paroxysm to return, and increases its violence; and at length, perhaps, an aggravated attack occurs which puts a period to the patient's existence. So seems to have died the late John Leech.

Prognosis.—The danger is in proportion to the nature and degree of the organic lesion on which the disease depends. Sir John Forbes found that of sixty-four recorded cases of angina forty-nine died, almost all of them suddenly.

Treatment.—The indications for the treatment of *angina pectoris* are to be found in a study of the lesions on which the paroxysms are found mainly to depend. Medicine can do little more than mitigate the severity of an attack; and this is generally best done by diffusible stimulants, such as *brandy, ether, chloroform, ammonia, chlorodyne.* Alcohol in small doses often repeated, *sesqui-carbonate of ammonia* in doses of *three* to *five* grains, the *muriate of ammonia* in doses of from *ten* to *twenty* grains, have each purely powerful stimulant effects. Hot bottles and sinapisms should be applied to the feet. The bowels may require to be rapidly and efficiently acted upon.

CYANOSIS.

LATIN EQ., *Cyanosis;* FRENCH EQ., *Cyanose;* GERMAN EQ., *Cyanose*—SYN., *Blausucht;* ITALIAN EQ., *Cianosi.*

Definition.—*A peculiar blue condition of the skin symptomatic of various malformations or derangements of the heart and great vessels, so that a small portion only of the blood is subjected to aëration in the lungs.*

Pathology.—The blood corpuscles absorbing oxygen in the lungs convey it through the circulation to every part of the body, and thereby render the metamorphosis of tissue possible. If this absorption of oxygen in the lungs by the blood-corpuscles is for any reason stopped or diminished, *arterial* blood retains the properties of *venous* blood, and the condition termed *cyanosis* arises.

In its *minor degree* it is associated with various forms of cardiac and pulmonary derangement having the effect of ob-

structing the flow of blood in the veins of the lungs and of the system generally. The chief of these is constriction or partial obstruction of the pulmonary artery, combined with systemic venous engorgement. In cases of cyanosis the skin is usually thin, the capillaries abnormally large; hence, when obstruction to the pulmonary and systemic venous circulation causes imperfectly aërated blood to flow throughout the system—and still more so in cases when, in consequence of some congenital malformation, a small portion only of the blood is subjected to the aërating influence of respiration—a dark, dusky, more or less livid hue is imparted to the skin.

In its more severe form the condition is usually associated with such a malformation, disease, or injury of the heart or great vessels, as permits venous and arterial blood to mix, and after mixture to be so distributed to the systemic capillaries. A patulous condition of the *ductus arteriosus*, an open *foramen ovale*, a deficiency of part of the *septum* of the ventricles, a heart formed of one ventricle and one auricle only—the aorta and pulmonary artery rising from a common trunk—are amongst the usual conditions which lead to cyanosis.

It is usually, therefore, a congenital affection, and the physical signs vary with the precise condition of the heart and arteries to which the cyanotic discoloration is due. The action of the heart is usually more forcible than in health, and hypertrophy and dilatation of the right ventricle are almost always present. Deficiency of animal heat is also a constant phenomenon.

The causes of cyanosis are arranged by Vogel into two groups:—(1.) In the one group the supply of oxygen to the blood, and consequently the oxydation of the blood-corpuscles, is prevented by derangements of the respiration or circulation (*i. e.*, by causes which are external to the blood); (2.) In the other group the blood-corpuscles lose the property of absorbing oxygen. This occurs in some cases of *pyæmia* and the last stage of *pulmonary tuberculosis*. There seems to be a diminished capacity of the blood-corpuscles to redden themselves.

Malformations of the Heart may be here simply enumerated. They may be arranged under the following heads:—

1. *Misplacements of the heart* (*Ectopia Cordis*) occurring congenitally, as in cases of transposition of the viscera. For an account of all that is known on this subject, the reader is referred

to a paper by Professor Allen Thomson in the *Glasgow Medical Journal* for July, 1853; and to the *Lancet* of August 8, 1863, where the condition is described by Professor W. C. Maclean as having been recognized during life. For an excellent report on cases of *ectopia cordis*, see *Pathological Society's Transactions*, vol. vi., p. 98.

2. *Congenital deficiency or absence of the pericardium*, in which the heart is, as it were, naked, and lying in one cavity with the lungs (*Path. Society's Trans.*, vol. iii., p. 60; vol. vi., p. 109).

3. *Arrest of development of the heart at an early period of fœtal life*:—as in hearts with one auricle and one ventricle; with imperfect separation of the ventricles; cases of contraction or absence of the pulmonary artery, the aorta arising from the right ventricle, and the *septum* of the ventricles imperfect. In cases of imperfect *septa*, the aorta sometimes arises from the two ventricles. Sometimes the ventricular *septum* is wholly absent.

4. *Premature closure of fœtal passages—the foramen ovale and ductus arteriosus—malformations causing changes which ought not to ensue till after birth.* The consequences of such premature results are chiefly cyanosis, combined with imperfect dilatation of the branches of the pulmonary artery.

5. *Irregularity of the valves, and origins of vessels, which may not in the first instance interfere with the functions of the heart, but which are apt to lay the foundations of disease in after-life.* Examples are to be seen in cases where two aortic valves occur; where there is fusion or union of two of the valves; where there is transposition of the aorta and pulmonary artery, both auricles opening into the left ventricle (l. c., vol. vi., p. 117). Excess of pulmonary valves (l. c., vol. iii., p., 301; vol. iv., p. 102). (See *College of Physicians' Nomenclature*, p. 239.)

The **treatment** of malformations which are associated with cyanosis is mainly preventive of dyspnœa and palpitation, by the avoidance of fatigue and mental excitement, the maintenance of temperature, and especially by a nourishing diet and warm clothing.

PALPITATION AND IRREGULARITY OF THE HEART.

LATIN EQ., *Palpitatio et tumultus cordis;* FRENCH EQ., *Palpitations;* GERMAN EQ., *Herzklopfen und Unregelmässigkeit in der Herzthätigkeit;* ITALIAN EQ., *Palpitazione ed irregolarità del cuore.*

Definition.—*Disturbances of the action of the heart unconnected with organic mischief.*

Pathology.—The interest which attaches to this subject consists in the difficulty which sometimes exists in the recognition of *functional* as distinguished from *organic* disease; and the fact that great uneasiness and distress, both mental and bodily, result from the occurrence of functional disturbance, while its persistence is apt to induce organic disease. Death has resulted from simple functional disturbance (GRAVES).

The forms of functional disturbance which simulate organic disease of the heart are palpitation, fluttering, or a "rolling" action of the heart, sometimes associated with a valvular murmur and irregularity, feebleness, or altered rhythm of the heart's action. The causes of such phenomena are common to adolescence and middle adult life, and are most frequently traceable to excessive mental exertion and sedentary occupation; great anxiety and strong mental emotion; nervous exhaustion from various causes —such as uterine irritation, excessive venery, masturbation; the influence of poisons on the heart's action—such as that of tobacco, spirits; *gout, rheumatism;* derangements of the stomach and liver, characterized by the existence of flatulence and acidity (FULLER).

Exaggeration of the functional acts is characteristic, and the general symptoms indicate great distress, a sense of fulness and of deep oppression in the *præcordial* region, pain, breathlessness, and tendency to faintness. Frequent giddiness, with pain, heat of head, singing in the ears, flushing of the face, coldness of the extremities, are associated with forms of dyspepsia characterized by excessive flatulence, acid eructations, restlessness at night, depression of spirits, and mental anxiety as to the nature and probable issue of the disease;—all contribute to induce and maintain the functional disturbance of the heart (FULLER).

The characteristics of palpitation, as due to functional and organic causes, have been already indicated (see pp. 628 and

629); and repeated examinations of the patient are necessary to arrive at a just conclusion.

Perversions of rhythm have sometimes been attributed to functional disturbance merely; but when altered cardiac rhythm is not merely of temporary duration, but is attended with giddiness, faintness, or actual syncope, it is more likely (in the absence of positive knowledge as to the state of the heart's tissue) that such altered rhythm is due to organic or textural degeneration. In every case examined by Dr. Fuller he has found it to be so.

Section III.—Morbid Conditions of the Great Blood-Vessels and of the Blood.

AORTITIS.

Definition.—*Inflammation of the aorta characterized by pains, with local pulsation along the course of the aorta or under the sternum, associated with rigors and pyrexia, with apnœa and great palpitation of the heart.*

Pathology and Symptoms.—Mere redness of the artery, without any alteration of the inner membrane, may be simply cadaveric, owing to imbibition of blood-pigments. The *post-mortem* appearances of true aortitis are,—great vascularity, and especially of the *vasa vasorum*, a thickened and pulpy state of the inner and lining membrane, which, having lost its glistening aspect, has a villous, rugous, or granular aspect. Lymph may be seen on its surface, and slight excoriations or superficial ulcerations may be observed here and there. The elasticity of the vessel no longer exists, so that rupture takes place very readily. Local pulsation is extremely energetic; and as we know that in cases of inflammation of the carotid there is pain when the vessel is pressed upon, so pain in an inflamed aorta may be aggravated by motion. A murmur is appreciable—a loud, rough, systolic bruit, due to the passage of blood over the rugous and unpolished state of the inner membrane (Gendrin, Parkes). In the case related by Dr. Parkes it was heard over the third dorsal vertebra, down into the lumbar region (*Med. Times*, Feb. 23, 1850).

Causes.—Rheumatism, gout, and syphilis seem to have a decided influence in the development of this disease.

Treatment.—Leeches should be applied freely over the course of the vessel; and large doses of opium, or of *ether* with *chloroform*,

may be given to relieve pain and dyspnœa. Iodide of potassium, or colchicum, may also be indicated.

ANEURISM OF THE AORTA.

Definition.—*A circumscribed dilatation of some portion of the aorta, consequent on lesion of some of its coats.*

Pathology and Symptoms.—They most frequently arise from the ascending portions of the arch, and seem to spring most frequently from those parts of the vessel against which the current of the blood is most forcibly directed. The effects produced vary with the seat and the size of the tumor; and aneurism is generally one of the most distressing and puzzling of thoracic diseases.

Aneurisms opening upon mucous surfaces, especially upon the air-passages, are generally attended with small and irregularly-repeated hæmorrhages. The persistence of these trifling amounts of blood in the expectoration justifies suspicion of aneurism, in the absence of any other circumstances to account for it. Tumors, such as are caused by aneurism, often give rise to such symptoms as are suggestive of laryngeal disease; therefore, in all cases, the larynx ought to be examined on the one hand by the laryngoscope, and on the other, a physical examination of the chest should be made for the signs of an aneurism or tumor. The frightful and agonizing dyspnœa is generally due, in cases of aneurism, to implication of the recurrent laryngeal nerve, and not to ulceration or disease of the larynx.

In some cases of thoracic aneurism the dyspnœa may have a spasmodic asthmatic character, without any tendency to laryngeal spasm, associated with the persistence of small hæmorrhagic expectorations. In such cases the aneurismal tumor presses upon the bronchi towards the roots of the lungs, evidence of valvular disease and of pneumonia being absent.

In another class of cases paroxysmal sufferings, in the form of *angina pectoris*, have their origin in the interference by an aneurism with the thoracic nerves, or with the venous circulation in the heart itself.

The variable character of the hæmorrhage, and the remarkable intermissions of such hæmorrhages from aneurismal sacs, are symptoms of thoracic aneurism which are now only beginning to

be appreciated in their proper light, especially since the case of Mr. Liston drew the attention of the profession to them. Hæmorrhage may occur in one of the three following forms:—(1.) A frothy bronchitic sputum streaked with blood; (2.) A rusty sputum, very like pneumonia, but usually more abundant, more frothy, and less viscid; (3.) A deeply dyed purple or brownish-purple sputum, like the so-called "prune juice" expectoration characteristic of the third stage of pneumonia, and of certain forms of pulmonary hæmorrhagic condensation from valvular disease of the heart; (4.) Any of the preceding forms of hæmorrhage alternating with small discharges of pure, unmixed, but generally imperfectly coagulated blood. The bronchitic varieties of sputum, either stained or streaked in different proportions with blood, occur chiefly in tumors pressing on the trachea and larger bronchi, and not producing consolidation of any part of the lung; while the "prune juice" sputum occurs when the lung is directly involved in the tumor, (W. T. Gairdner's *Clinical Medicine*, p. 454, *et seq.*)

Permanent contraction of the pupil on the affected side is in some cases another sign of aneurism, which Drs. Reid, Gairdner, Ogle, and others have clearly demonstrated.

The co-relation of the symptoms, as possibly indicating thoracic aneurism, is the main point for attention; and in addition to those noticed, raucous voice and aphonia are found sometimes associated with thoracic aneurisms. Undue pulsation, dyspnœa at intervals, oppression at the præcordial region, with difficulty of swallowing solid food, are, in combination, significant of aneurism.

The sphygmograph is calculated to yield important aid to diagnosis (SANDERSON, ANSTIE, and FOSTER), by showing in the pulse-trace the modifications in the movement of the blood produced by the diseased condition. These modifications are intimately dependent on the seat of the tumor, its size relatively to the vessel with which it is connected, and the elasticity of its walls (FOSTER).

The more important modifications may be considered:—*first*, As to aneurism so situated on an artery that the pulse can be observed on the vessel below the tumor; and *secondly*, Aneurisms affecting the aorta. The beat of an artery, when carefully felt below an aneurismal tumor which implicates it, is found to present unusual characters: it is weakened, and generally retarded. The sphygmograph shows that modifications of

the pulse occur both in its form and force. In the pulse-trace collected from an artery below an aneurism, the movement of the blood in the vessel approaches rather to that which is normally seen in the smaller arterial branches. The vertical line of ascent disappears; and often this line approaches in length that of descent. Thus we have a more feeble pulsation given to the finger; and, as the summit is slow to occur, there is a sensation of apparent retardation felt in addition. The following figures (34, 35) represent the modifying influence of an elastic aneurismal sac on the pulse-trace of the left radial artery (Dr. B.

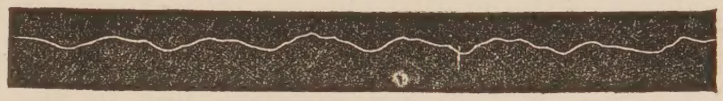

Fig. 34.*

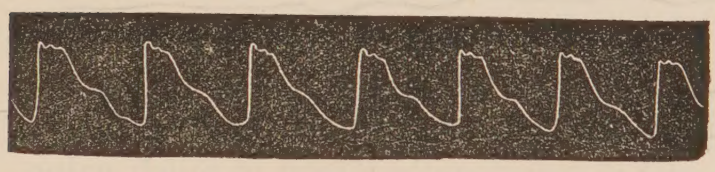

Fig. 35.†

Foster). There was an aneurism of the left subclavian artery within the thorax. The right pulse shows the trace of another lesion under which the man laboured—namely, insufficiency of the aortic valves. So characteristic of the conditions in this case were the above traces, that they alone enabled Dr. Foster to arrive at the diagnosis to which ordinary means had led those watching the case. At the time when the above traces were taken, the tumor was evidently large and very elastic—hence the great modifications in the pulse-form produced.

In *aneurisms of the aorta*, very much less striking indications are afforded by the pulse-trace. Nevertheless, Dr. Foster's experience leads him to believe that the position of a thoracic aneurism may be indicated by the pulse changes—*e. g.*, when a small aneurism or even a considerable dilatation of the ascending aorta exists, the pulse of the right radial is generally reduced in size, when compared with the left (Dr. Foster's *MS. Notes*). The indications pointed out by Marey are,—(1.) Modifications in

* Left radial. † Right radial.

the force of the pulse; (2.) Modifications in the intensity of the dicrotism; and (3.) The existence of a constant difference in the pulse-form of the two radial arteries.

(1.) The force of the pulse, according to Marey, is seldom much diminished—a character of small value in diagnosis. The causes of this want of change in force reside probably (*a.*) In the small size of the tumor relatively to the volume of the aorta; (*b.*) In the fact that the sac is sometimes not placed in the direct route of the blood, but communicates with the vessels, and thus has a much less transforming effect upon the blood-movement than a tumor which must be traversed by the current; and (*c.*) In the thickness and slight elasticity of the wall of the sac often found in aneurisms in this situation. The force of the pulse, too, in these cases is altered very often, not in one artery alone, but in the vessels of both sides of the body; and, on the other hand, it must be remembered that the tumor, by compressing the orifice of one of the branches of the aorta, may cause a peculiarity in the radial of one side.

(2.) The modification in the dicrotism may exist in one or both radial pulses, and is occasionally a sign of much value.

In aneurisms of the descending thoracic aorta the dicrotism proper is often much increased in both pulses, but especially in the right, while the left radial is smaller than the right. This latter fact may be explained by the relation of the innominate and left subclavian to the blood current, the former of which receives the full force of the blood discharged from the ventricle; but the current has to deflect itself slightly to reach the left subclavian, and is partly drawn into the descending thoracic aorta where the aneurismal sac gapes to receive it. The dicrotism is increased in these cases by the contraction of the aneurismal sac (Dr. Foster).

(3.) The presence of a constant dissimilarity in the pulse-traces of the radial arteries is the most valuable sign in the diagnosis of aortic aneurisms. In many cases the finger can perceive a want of parallelism in the beats of the radials, but often this is too slight to be detected by the finger. It sometimes shows itself in the trace by a slight difference in the dicrotism only; at others, the difference in form is more evident. When the tumor is so situated that it can be handled, we can gain valuable evidence as to its nature by observing the changes in the tension of the

arteries, produced by its alternate compression and relaxation (Dr. B. Foster).

Causes.—The causes of thoracic aneurism are exceedingly obscure; but there is good reason for believing that the morbid constitutions associated with *gout, rheumatism,* and *syphilis* are the circumstances under which aneurisms are most apt to be developed, the elasticity of the vessel being impaired by structural changes. With regard to the influence of syphilis, I may here observe that I have dissected, during the past four years (at Fort Pitt and at Netley Hospitals for invalids), twenty-six bodies of soldiers, in each of which a distinct history of syphilis was present, associated with unmistakable syphilitic lesions; and of these twenty-six cases, *seventeen* had the coats of the thoracic aorta impaired by characteristic changes—changes which are uncommon at an early period of life, and which I have every reason to believe are due to syphilis. The changes are obvious from cicatricial-like loss of substance of the inner coats, small local dilatations of the artery, and in several cases aneurismal expansions, one as large as an orange, which proved fatal. A characteristic case of aneurism of the thoracic aorta resulting from syphilis is also recorded by Assistant-Surgeon Alfred Lewer, in the *Medical Report of the Army Medical Department* for 1862, p. 512. Hereditary transmission of aneurism has been noticed by Dr. Fuller.

Treatment.—Local bleeding is useful when there is pain and tenderness over the aneurismal sac; and general blood-letting may be useful if the circulation is excited, and the patient be of full habit, but not on the principle advocated by, or ascribed to, Valsalva. Of all remedies, *digitalis, aconite,* and *veratrin* are the most useful in tranquillizing the action of the heart. They tend to render the circulation slower, without deranging the action of the stomach. The deposition of fibrin from the blood is more prone to take place when the circulation is "slowed;" indeed, it is the principle of treatment in the cure of aneurisms by pressure. The current of blood is not stopped, but is simply rendered more slow, so as to have an amount of stagnation of blood in the sac favouring the separation of fibrin and its coagulation. A diminution of from *ten* to *fifteen* pulsations of the heart in the minute will thus greatly tend to the filling of the sac with coagula (Fuller).

A most interesting paper has just been published in the

Medical Report of the Army Medical Department for 1862 (p. 472), from the pen of Joliffe Tufnell, Esq., advocating the treatment of aneurisms of the thoracic and abdominal aorta on the principle here enunciated, of "slowing" the circulation. His treatment consists of restricted diet and perfect rest in the horizontal position, for periods varying from eight to thirteen weeks, combined with the employment of such remedies as may be necessary for special ends. The horizontal posture must be *strictly and absolutely maintained,* in a light and cheerful airy room, into which the sun shines, and from which the patient may be able to have as cheerful a view as possible out of the window.

The diet must be confined to *three* meals, served at regular intervals, and restricted to the following in kind and in amount:—

Breakfast—Two ounces of white bread and butter, with two ounces of milk or cocoa.

Dinner—Three ounces of broiled or boiled meat, with three ounces of potatoes or bread, and four ounces of water or light red wine.

Supper—Two ounces of bread and butter, and two ounces of milk or tea.

These diets should make in the aggregate *ten ounces of solid and eight ounces of fluid food in the twenty-four hours, and no more.* The object of the special diet is to maintain life on as little food as possible without inducing restlessness, as in some irritable constitutions; but if such restlessness should occur, a little more food may now and then be allowed. *Anodynes, aperients, narcotics, sedatives,* and *tonics* are also useful aids in the management of the case. Of anodynes, Mr. Tufnell regards *lactucarium* as the most valuable, given in the form of a pill, either by itself or combined with *humulin* and *hyoscyamus.* Mr. Tufnell's practical suggestions in detail will well repay a careful study, and merit publication in a more accessible form than in a "*blue-book.*"

Tracheotomy may prolong life in some cases where *stridor* exists, if the laryngeal symptoms are the source of immediate danger (W. T. Gairdner).

PYÆMIA.

LATIN EQ., *Pyæmia;* FRENCH EQ., *Pyohémie;* GERMAN EQ., *Pyaemie;* ITALIAN EQ., *Piemia*—Syn., *Piemasia.*

Definition.—*A febrile affection resulting in the formation of abscesses in the viscera and other parts, and usually associated with phlebitis.*

Pathology.—There are febrile conditions in which the blood is materially disturbed in various ways, and which tend to complex forms of lesions in many parts, and especially to multiple centres of inflammation, with a great tendency to suppuration. A number of morbid processes, having many elements in common, of great practical importance, of frequent occurrence, and dangerous to life, have been described of late under the very various names of—(1.) *Simple pyogenic fever* (JENNER); (2.) *Acute purulent diathesis* (TESSIER); (3.) *Septic poisoning of the blood* (the septicæmia of VOGEL); (4.) *Systemic infection* (the ichoræmia of VIRCHOW); (5.) *Putrid fever;* (6.) *Pyæmia;* (7.) *Phlebitis.*

The multiplicity of views and great uncertainty which still pertain to many points regarding the pathology of the phenomena comprehended under these various names, make it necessary to write with doubt and some hesitation; more especially as the details into which the topics may be carried which are embraced under this head are daily becoming more and more extensive. The aim of this account, therefore, is to put the student in possession of the more important bearings of the subject, in relation especially to pyæmia, phlebitis, and the phenomena of embolism.

The first two—namely, the *pyogenic fever* of Jenner, and the *purulent diathesis* of Tessier—are characterized by the occurrence of *multiple abscesses.* The remaining *five* of the morbid states which have been enumerated have been recently classed by Virchow, Bilroth, and others, under the common name of "metastatical dyscrasiæ." In them there is evidence of the occurrence of secondary or metastatical inflammation—a tendency to multiple centres of inflammation, to "multiple abscesses," and to suppuration in various parts of the body. Associated also with these lesions is the occasional formation of *clots, thrombi, plugs,* or *embolia,* the occurrence of *thrombosis,* or of *embolism,* or of

phlebitis, softening of the minute tissue of visceral organs, such as the *brain*, the *lungs*, the *heart*, the *liver*, or the *kidney*, or of *gangrene* of the extremities.

Pyogenic Fever.—In the Gulstonian lectures delivered at the Royal College of Physicians in London, for 1853, Dr. William Jenner directed particular attention to some lesions which are apt to follow immediately after the termination of acute specific diseases, such as scarlet fever and the like. The simplest form of these lesions consists in the formation of several (or multiple) abscesses of small size in the subcutaneous connective tissue, especially of the scalp, chest, loins, legs, or arms, accompanied by more or less febrile disturbance. Such multiple abscesses are (on circumstantial evidence only) presumed to owe their origin to a diseased condition of the blood—to represent what is popularly known as "the dregs of the fever"—as the media by which something unwholesome is ultimately evacuated—or as a *crisis* in which the specific affection terminates. Sometimes, on the other hand, these abscesses are accompanied by severe constitutional disturbance, and instead of being superficial, are deeply seated, either in the connective tissue or into the cavities of the joints, and in rare cases even into the serous cavities of the cranium, abdomen, or thorax. Cases of all these varying degrees of severity are observed, which seem to differ only in the more or less wide diffusion of the local affections. Such cases are found to be in close alliance with those diseases in which purulent discharges issue at the same time from the mucous passages, and to that chronic cachexia in which the least scratch or abrasion of the skin tends to "fester" and not to heal. This condition of the blood and system was first recognized and described by Tessier, in 1838, under the name of the "purulent diathesis," or "a tendency to suppuration in the solids and coagulable fluids."

In the cases which are recognizable as belonging to this class of diseases, the febrile disturbance seems to be established before any local lesion is set up; and the morbid condition of the blood thus came to be looked upon as "a primary substantive affection," because it seemed to lead to a "fever" followed by these lesions.

Cases of this morbid condition are related by Dr. Jenner in the *Medical Times and Gazette* for May 7, 1853; and they may be compared with a case of multiple abscesses in and about the joints, associated with rheumatism, and described by Dr. Bennett in his

valuable *Principles and Practice of Medicine*, p. 803, under the name of *ichorœmia* or so-called *pyœmia*.

The subcutaneous tissue and the joints are the most frequent seats of these abscesses; much less frequently the lungs or other viscera, which in the following class of cases are the most frequent seat of the multiple abscesses. Here we have an important distinguishing character between the two classes of diseases—namely, that the abscesses of pyogenic fever seem to be developed out of a *constitutional* state of general ill-health, whereas those which are about to be considered may be regarded from the beginning as due to a blood-poison, or other causes of irritation set up from external sources. In the former case the cause of the disease seems to be *constitutional;* in the latter, *specific*. Hence the College of Physicians places pyæmia among the general diseases, Class A (see vol. i., pp. 183 and 185). Again, the class of lesions associated with pyogenic fevers is still further distinguished as of one kind by the following characters:—(1.) The abscesses are not consequent upon the pre-existence of any abscess, ulcer, or clot, or plug in the blood-vessels; (2.) There is no evidence of the formation of pus or of fibrinous debris in the veins, nor of the passage of that fluid into the blood, and the establishment of (so-called) *pyœmia;* (3.) The symptoms, and still more the *situation*, of the disseminated abscesses differ in cases of *pyogenic* fever from those which occur in the so-called cases of *pyœmia* or *phlebitis*.

It is with typhus or typhoid fever only that pyogenic fever is apt to be confounded; but from this fever it is distinguished by the activity of the febrile symptoms at the outset, the early delirium, the absence of eruption, and the rapid formation of the numerous centres of suppurative action. The closest alliance, pathologically, of pyogenic fever seems to be with phlegmonous erysipelas.

Metastatical Dyscrasiæ, under the generic name of pyæmia, have attracted considerable notice from two points of view :—

(1.) In connection with various conditions of the veins, to which the name of "phlebitis" has been given; (2.) In connection with various changes in the blood itself, and more especially of late years, as described by Virchow and Bennett, under the name of "leucocytosis" (*ante*, p. 70). Several scientific questions associated with each of these views are still the source of controversy.

Pyæmia literally means a condition in which—(1.) There are pus-cells in the blood; but the expression has now come to imply—(2.) That the blood is altered throughout the whole system by the poisonous action of putrid animal substances, in the form of gases, fluids, or solid particles, which so disturb its relations with the living tissues as to induce coagulation of the fibrin of the blood in some part, during life, within the blood-vessels, associated with fever, and the formation of local abscesses in one or more of the viscera and other parts, and usually accompanied by *phlebitis*.

This latter signification of pyæmia has been in some measure forced upon us, because it is found impracticable to say when any given specimen of blood is full of white blood-discs, or of pus-corpuscles. It was once supposed that pus could be absorbed *as pus*, and be conveyed away in substance; and that two results might follow such an event: either—(1.) That the pus would be passed off by the urine or the fæces as an excretion, but still in the form of the original pus; or, (2.) That the phenomena of pyæmia would be the other alternative.

Now we know that neither of these results ever takes place. Pus, *as pus*, is never taken up or absorbed into the system. Its fluid part only may be absorbed by veins or lymphatics; but the solid portion remains as a thick, inspissated, or concrete mass; and thus the absorption or disappearance of an abscess sometimes gives rise to such "cheesy" products as are described by the name of "tubercle;" and which may subsequently induce ulceration, as a foreign body would. Again, pus may be completely absorbed; but only after the cells have been reduced to the state of milkiness by fatty degeneration, and become converted into an emulsive mass—a kind of milk, composed of water, albuminous matter, fat, mucus, cholesterine, sulphates, lactates, and the like, and in which also sugar may be present.

The composition of pus varies considerably; and the conditions under which it varies have not yet received the attention which its importance demands. It seems to vary with the locality whence it comes, and with the circumstances under which it is formed. The pus of a *pulmonary vomica* differs from that of a *psoas abscess*, and that again from the pus of a *mammary* or *hepatic abscess;* and so also the pus of *syphilis* differs from that of *small-pox*. The very large and variable amount of

organic elements which pus contains renders it also extremely liable to change; and the products of decomposition of the elements of which pus is composed are extremely various and diffusible—*e. g.*, ammonia, gas products, and salts, leucine and tyrosine; the acids also of the butyric group, as well as formic acid. Another fact to bear in mind is the "spontaneity" which they (and especially albumen) undergo the process of putrefaction, without any *apparent* co-operation of other matters, and solely by the influence of atmospheric agents; and as constant products of such putrefaction there are always to be found *carbonate* and *butyrate*, and *valerianate of ammonia, sulphide of ammonium, leucine*, and *tyrosine*. By putrefaction, and under the influence of impure air especially, pus may undergo an acid or alkaline fermentation. The former is rare; but when it does occur, there are developed volatile and fixed fatty acids, such as *butyric acid* and *margaric acid*.

Unhealthy pus more commonly tends to become alkaline without becoming acid. It evolves ammonia and hydrosulphate of ammonia. The corpuscles soften and run together, nuclei disappear, and the whole becomes a mass of granules, as is the case in hospital gangrene (as I have repeatedly observed), which are probably capable of re-absorption.

Pus essentially consisting, as it does, of cells in a pus-serum varying in size from $\frac{1}{2000}$th to $\frac{1}{3000}$th of an inch, has only one way in which its cells can find their way into the blood—namely, by the perforation of a blood-vessel by an ulcer or a puncture. An abscess close to a vessel may open into it; but the result is harmless if the pus is fresh and healthy. The passage of pus into lymphatic vessels is still more easy when such vessels run into open abscesses. But then lymphatics do not empty their lymph into blood-vessels (with one exception) till they have elaborated such lymph in the lymphatic glands; and from the nature of gland-structures it is known that no pus-corpuscle, as such, can pass a lymph-gland. Such glands not only *filter* mechanically, but are living absorbents of some constituents only with which they come in contact, and amongst others, no doubt, the constituents of the pus-fluid without the actual pus-corpuscles, whose debris would simply be retained within the glands. Irritation of these glands leads to proliferation of the gland-cells, and subsequently to the passage into the blood, by the left jugular

vein, of cells which cannot be distinguished from pus—the colourless blood-cells. Under such circumstances nothing is easier than to demonstrate what seems to be pus. The presence of white or pus-like cells in the blood, by what we know of blood-formation, can be explained. We know that the blood gets these blood-cells —(1.) After every meal we take; (2.) By irritation of lymphatic and other glands from cachexia; (3.) With the advance of pregnancy and splenic enlargement. In scrofula, typhoid malaria, and cancer, they also abound.

The existence of pus in the blood cannot therefore be demonstrated as the term "pyæmia" has hitherto been understood to mean; and now, therefore, when the term is used, it is meant to imply that an unknown matter, derived apparently from the spontaneous decomposition of some kinds of purulent or albuminoid substances, has mingled with the blood—has poisoned that circulating fluid, or has so altered it that it tends to coagulate in the vessels during life, and has given rise to various secondary phenomena about to be noticed.

"Pyæmia," as a specific original entity—as a result of absorption of pus, as such—is not now believed in; and any evidence of such an event daily becomes less and less obvious. *Pyæmia* is now rather to be regarded as a collective name for many very different disease-processes, just as the essential phenomena of Bright's disease are brought about by several different morbid conditions of the kidney. It is especially necessary to distinguish between the following phenomena—namely, (1.) Leucocythæmia; (2.) Embolism, with resolution of clots, putrid decomposition, or gangrene; (3.) Absorption of putrid fluid, without embolism; (4.) Septic endosmosis of gases, independent of embolism.

To these phenomena (excluding those connected with leucocythæmia) Virchow has given the general term of "metastatical dyscrasiæ;" and amongst them *phlebitis* and *hospital gangrene* are too often fatal, as the result of operations under certain unhygienic conditions.

The phenomena presented by individual cases, capable of being classed under the head of *metastatical dyscrasiæ*, are sometimes so extremely varied that names have been given to diseases as well as to lesions which do not always have the same limit or range of significance.

Phlebitis.—The first set of phenomena to be considered are

those which accompany *phlebitis*. So long as pus-corpuscles, as such, were looked upon as the really noxious material, it was supposed that the tissue of a vein being inflamed, pus would be secreted from its inner wall, just as from a serous membrane. John Hunter has the credit of having suggested this; but he did so merely *as a query*, and subsequent writers adopted the suggestion without further examination and without evidence, (see Arnott "On the Effects of Inflammation on Veins," in vol. xv. of *Med.-Chir. Trans.*) Veins are exceedingly slow to inflame. When they do, the inflammation-changes begin in the connective-tissue, towards the outer parts of the vessel, and this even when irritant bodies are introduced into the cavity of the vein itself, (see Lee's "Essay," in vol. xxv. of *Med.-Chir. Trans.*) Mr. Lee introduced cotton wool into a portion of vein emptied of blood. The lining membrane remained unchanged; and the lesions commenced outside the vessel. There the pus formed, and thence the inflammation spread by the connective tissue, and simply by continuity. The generation of secondary multiple abscesses is attended by quite a different process. It was first shown by Cruveilhier that in the (so-called) inflammation of veins a clot of fibrin forms, and is always present, independent of any lesion in the vascular wall; and so he passed to the extreme belief that all inflammation essentially consisted in coagulation of blood in capillary vessels. The first great fact has now been quite substantiated—namely, that long before any visible effects of inflammation occur in the lining membrane of a vein, a clot of fibrin is formed at the part, and in this clot fluid comes to be produced, in all external appearances resembling pus. It must also be remembered that blood, when arrested in the vessels or extravasated out of them, coagulates at the ordinary temperature of the body—98° Fahr. (*Phil. Trans.*, vol. lxxxvi.)—a most valuable event in the cure of aneurisms. From this observation as a starting-point, Virchow has developed his very beautiful explanations of the various phenomena connected with phlebitis. Inflammation of veins as the cause of secondary inflammatory phenomena and of multiple abscesses is to be rejected. "Coagulation of the blood in the living vessels"—the formation of a clot or "thrombus" (*thrombosis*)—are the phenomena which attend the formation of multiple abscesses in the phlebitis of pyæmia. The impulsion or projection onwards of a coagulum-clot, or thrombus, or substance

detached from the walls or valves of the vascular system, is described under the name of *embolism.*

The coagula may travel in particles or larger masses from the veins to the heart, or from the heart to the arterial peripheric vessels.

Thus, on the one hand, *deposition* of morbid substances in various parts of the body distant from the heart is accounted for; and, on the other hand, results are explained on simple mechanical principles which hitherto have been obscure. Formerly many of the cases thus explicable would have been recorded as cases of "sudden death," or as "sudden retrocession of gout or rheumatism," or of "gout in the stomach," or "palsy of the heart."

All the cases, fully recorded, which illustrate the phenomena of so-called pyæmia or phlebitis, that I have ever examined, have shown that the affection essentially begins by a real coagulation of the blood at *some definite fixed point;* and this is the most obscure part, and the most difficult to discover, in the history of all the cases. But where this beginning is traceable, the history is exceedingly significant, as pointing to some sources of *local irritation,* which, by simple disturbance of the flow of blood, determines in some way its coagulation in the living vessels. The beautiful experiments of Professor Lister throw much light upon this subject ("Croonian Lectures," *Lancet,* Aug. 8, 1863). Some of the cases mentioned by M. Ribes, in 1825, occurring so far back as 1799, illustrate this point by morbid anatomy. In one case chilblains was the starting-point. Clots formed in the veins, and proceeded up even to the superior vena cava, into the right auricle and ventricle. In one of the most striking cases of this description recently recorded, "a venous clot of fibrin twenty inches long was found in the right auricle and ventricle (Druitt, *Med. Times and Gazette,* July 19, 1862). It showed such marks on its surface as clearly demonstrated its formation in a vein; and other circumstances pointed to the chief vein of the thigh as the site of the primary formation of this *clot* or *thrombus.* Œdema of the limb prevailed, and the disappearance of the œdema was associated with those sudden cardiac and other symptoms which indicated the passage upwards of this *coagulum* or *thrombus* to the heart, where it was found coiled up in the right auricle and ventricle. In cases of fracture of bone, of amputations, of

enlarged glands, of ulcer on the foot (imperfectly healed by scabbing), of open wounds, gunshot passing near vessels, and ulcers, we have great and many sources of irritation. The contiguity of these must be to more or less large veins, or to smaller vessels in or about these sources of irritation and disturbance.

"The immediate cause of pyæmia in any given case is, that some diseased part (which need not be an external wound) so affects the blood circulating through it, that this blood afterwards excites destructive suppuration in parts to which the circulation carries it—namely, commonly first in the lungs, or (in certain cases) liver and lungs, and later generally about the body" (Simon, *Public Health Report* for 1863, p. 60). In a case of which Mr. Simon was cognizant, and which is described by Mr. Bowman, fatal pyæmia in a young gentleman was apparently produced by an ulcerated state of the mitral valve of the heart. In another case Mr. Simon records fatal pyæmia produced by the penetration of pus from a small mesenteric abscess into the thoracic duct; and in the thirteenth volume of the *Pathological Society's Transactions* Dr. Bristow records several instructive cases, in which pyæmia complicated, *ab initio*, cases of *idiopathic necrosis unattended by external wound*. In no case, therefore, of swelled legs, enlarged glands, subacute inflammation, or hardening over the course of lymphatics or superficial veins, should the possibility of the mortal accident of *embolism* to the right side of the heart be overlooked; or the possible supervention of pyæmia.

Hospital Gangrene.—The influence of foul air, by inducing decomposition, the liberation of absorbable gases and other fluids also influence the blood passing the part, and bring about not only coagulation in living vessels, but also poisoning of the blood itself, as in hospital gangrene—"*a sloughing phagedena occurring endemically in hospitals.*"

Cases prone to Hospital Gangrene.—The disease shows a preference for those cases where cancellous bone-structure has been injured, as in compound fracture, or in the surgical procedures of amputation and resection; and all the more, perhaps, in proportion as the injured bone is large. The other cases in which it is apt to occur are those in which large vein-trunks have been involved in traumatic inflammation (as in gunshot wounds). Although the exact local changes are not well understood, yet very often they seem part and parcel of a process not simply

suppurative, but involving also much foulness of wound, putrefactive softening of fibrin and blood-clot, with the evolution of such products as have been already noticed, which, entering the circulation, establish the pyæmic state, and therewith usually more or less of putrid infection of the blood. Many phagedenic and gangrenous materials are inoculable from patient to patient (Thomson's *Lectures on Inflammation*, 1813, p. 484), and are so far *specific* diseases. All the forms of disease which come under the term *metastatical dyscrasiæ* having thus, in common, an intimate affinity with ordinary putrefactive processes, they ought provisionally to be regarded as general diseases, of the class A (see vol. i., p. 183), the respective *contagia* of which may arise in any putrefaction of wound-products; and when such diseases as erysipelas or pyæmia break out in an hospital, it shows that the ventilation is inadequate to remove the *traumatic* or other organic impurities generated in the wards. Thus there prevails an atmosphere which contains much decaying animal matter of the kind which wound-surfaces contain; and any *specific* change arising in this atmosphere, or on any wound-surface which exhales its excreta into the atmosphere, has peculiar chemical facilities for infecting other wound-surfaces within the range of atmospheric influence (see Simon's *Sixth Report on Public Health*, pp. 61, 62). Therefore, in ill-kept hospitals, wounds go on badly. They undergo certain characteristic morbid changes. Erysipelas frequently attacks them. They become to a large extent gangrenous and phagedenic, tending to putrefactions of effused or otherwise stagnant blood, to the re-opening of half-healed arteries and veins, to septic and suppurative infections of the system, generalized under the term *traumatic infection*, all of which are comprehended amongst the phenomena of *metastatical dyscrasiæ*.

Such, pathologically, are some of the causes which may determine the formation of clots either in large or in small vessels. But, after Cruveilhier's observations, the next link in the chain of evidence regarding the nature of *metastatical dyscrasiæ* was established by an arduous worker (then in the Army Medical Department, and since a distinguished Professor in the Royal College of Surgeons of England)—namely, Mr. Gulliver, formerly surgeon of the Guards. He showed that the puriform mass in the interior of clots does not originate in the wall of the vessel or clot, but is produced by transformation of the central layers of the clot—

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a transformation which may be imitated, as he did by a chemical process. Sir James M'Grigor communicated Mr. Gulliver's observations to the *Medico-Chirurgical Transactions*. Mr. Gulliver examined the clots microscopically, and found that the fluid was not pus; and to show that his observation was intimately connected with the observation of Cruveilhier, I found (by mere accident) that a remark on this point, in Mr. Gulliver's handwriting, exists on one of Cruveilhier's drawings contained in the Library of the Army Medical Department now at Netley.

This is the point from which Virchow starts in his interesting account of this subject—namely, the character of the contents of these clots—a puriform, but not a purulent substance (as Gulliver first showed), composed of granules chiefly. The question, then, immediately suggests itself, "What becomes of them?"

The next set of phenomena characteristic of the phlebitis of pyæmia and the formation of multiple abscesses, are those connected with the softening, disintegration, and breaking up of the thrombi or clots. Virchow was the first to demonstrate the results that ensue. He showed (1847) the embolic characters of certain products previously thought to be inflammatory in their origin (*e. g.*, white fibrine-like masses in the spleen, &c.); and he arrived at the following conclusions:—(1.) The occurrence of fibrinous plugs or clots in the pulmonary artery long before death is always secondary, where obstruction is independent of pneumonia, or other changes in the parenchyma. They are apt to arise in any part of the venous vascular system anterior to the lungs in the course of the circulation—*e. g.*, in the veins of the limbs, as in the case described by Druitt, already noticed; in the pelvic veins, as after the operation of ligature for internal piles, whence the clots are carried by the current of the blood to the right side of the heart, and thence into the pulmonary artery, and so to the lungs. Experiments on animals also supported his views. He introduced the pith of the elder tree, as well as animal substances, into the veins, and so produced violent pneumonias, commencing with inflammatory hyperæmia. These localized pneumonias extending, led to rapid deposition in the air-cells of fibrinous exudations, which became purulent, or the portion of lung gangrenous. As these changes advanced in the lung, pleurisy very soon was developed at the periphery—at first producing fibrinous coagulable exudation slowly over the affected portion of the lung; but

rapidly, as it progressed towards the other side of the chest, inducing watery hæmorrhagic exudation into both serous cavities. The pleura then became gangrenous, and finally gave way to pneumothorax. Such severe lesions may be completed in so short a time as five days. Previously, however, to these important demonstrations of Virchow, there had appeared the observations of Paget on the obstructions of the branches of the pulmonary artery, and the sudden mode of death to which they gave rise (*Med.-Chir. Trans.* for 1844); but the importance of Virchow's observations consisted in demonstrating the transportation of clots or plugs of fibrin or of blood from different parts of the venous vessels to the heart. Such plugs are known also to be sometimes arrested in the liver, giving rise to so-called metastatic abscess; and while those plugs which find their way to the lungs have their origin in any part of the venous periphery, those which find their way to the liver originate either in the *portal venous system* or in those veins round the *rectum, prostate, vagina,* or *uterus,* which (communicating alike with the *systemic veins* and with the *inferior mesenteric veins* of the *portal system*) may induce multiple centres of inflammation, and abscesses in the liver as well as in the lung (*Med. Times,* January 18, 1862). Simultaneously with Virchow's, or a little after his observations, we have the observations of the late Dr. Kirkes on "The Detachment of Fibrinous Deposits from the Walls of the Heart" (*Med.-Chir. Trans.*, vol. xxxv., for 1852). Two most instructive cases of this kind have been recently related by Dr. Goodfellow, in which extensive arterial obstruction, gangrene of the lower limbs, and death, followed the separation of cardiac vegetations, (*Med.-Chir. Trans.*, vol. xlv.) Thus it was gradually proved, that as clots occur in the veins, so they also may occur in the heart's cavities; and next in the history of this interesting subject the connection is established between *peripheral clots* and *cardiac clots,* and how far the softening of these clots or *thrombi* may give rise to the phenomena indiscriminately described as *pyæmia* or *phlebitis.*

Secondary disturbances—not so much by the softened mass as by the detachment of larger pieces, and just so large as to be arrested in vessels more or less remote from the seat of the original clot—are known to occur: and on the advance of clots and their debris from small into large vessels, bits break off and

flow on into the stream of blood. Clots in peripheral veins, however small, are thus the sources of great danger. As a rule, they lead to secondary and multiple deposits and abscesses in the lungs; and it is chiefly differences in the size of the capillary vessels which determine their ultimate locality, where they act as any foreign body would. The debris of clots, and large cell-elements from clots, in the mesenteric veins, and from ulcers of intestines, passing through the liver capillaries and proceeding to the lungs, where they are arrested, illustrate this. The lungs have the smallest capillaries of all. They average from $\frac{3}{10000}$ths to $\frac{50}{10000}$ths of a line (scarcely sufficient to let pass a white cell of blood or of pus, which on an average measures $\frac{44}{10000}$ths of a line), whereas the liver capillaries have a much larger range—namely, from $\frac{20}{10000}$ths to $\frac{100}{10000}$ths of a line.

Puerperal Fever.—Virchow regards the metastatic abscesses in the lungs, one and all, as embolic, and founds much on the facts connected with puerperal fever—"*a continued fever, communicable by contagion, occurring in connection with childbirth, and often associated with extensive local lesions, especially of the uterine system, peritonitis, effusions into serous and synovial cavities, phlebitis, and diffuse suppuration.*" He observed that cases of puerperal fever with multiple abscesses in the lungs had always thrombi or clots in the pelvic vessels; whereas in those cases where the lymphatics only were inflamed there were no multiple secondary abscesses.

The position of embolia in the pulmonary artery induces more or less instantaneous asphyxia. Paget's, Richardson's, and cases recorded by other observers, of more or less sudden death, illustrate this; so also do some cases of gangrene of the lung. If death does not at once occur, the clot in the vessels of the lung becomes broken up. Its fragments pass to the left side of the heart, if not expectorated, and clots are induced there. The phenomena of embolism from the left side of the heart are then apt to be expressed by gangrene of distant parts, as of the limbs, softening of brain, or loss of vision. Gangrene, or decomposition of puriform matter, occurs most actively in the lungs, because here the process is brought most intimately in contact with the outer air; and here, therefore, the influence of impure air becomes so much more injurious; but cases of arterial embolism developed

in this way (*i. e.*, primarily in the lungs) are not as yet known to be numerous.

The sources of embolism therefore imply some knowledge of the circumstances under which the blood spontaneously coagulates, or permits the separation of its fibrin to take place in the living vessels. In a pathological point of view the following events in their order may be regarded as the most important:—(1.) Local disturbance to circulation; (2.) Stoppage or retardation of the blood-stream—*failure of heart's action;* compression or constriction of the blood-vessels; (3.) Cohesion of red or white blood-cells into a mass, as a result of diseased artery.

The sources of local disturbance are,—(1.) Irritation of the outer coats of the blood-vessels; (2.) Changes which take place in them; (3.) The formation of a clot; (4.) Changes take place in it, and it then furnishes ample materials for further mischief; or (5.) Organization of the clot takes place, when it is converted into connective tissue, and generally remains harmless.

Arterial Emboli are observed as a consequence of—(1.) Gangrenous phlebitis of the pulmonary veins or of pulmonary tissue (*rare*); (2.) Organic affections of the aortic or mitral valves on the left side of the heart, fibrinous concretions, and warty excrescences (*common*); (3.) Atheromatous disease of the inner membrane of the artery. A first obstruction is generally followed by others—multiplicity and complexity of lesions being one of the characters of the disease. The most frequent sites of embolic arterial thrombi are,—(1.) Arteries in the fissure of Sylvius; (2.) Internal carotids; (3.) Arteries of the lower and upper extremities; (4.) Splenic and renal arteries; (5.) External carotid and mesenteric arteries.

The symptoms in sequence characteristic of the lesion may be noted as follows:—(1.) Valvular disease; (2.) After exertion, there occurs (3.) Palpitation, with the cessation of which (4.) The pulse disappears in the affected artery below the site of the plug—*e. g.*, in the *radial*—if the plug is arrested in the *brachial*, with pain in the hand of that side. These phenomena are all due to the sudden separation of fibrin from the warty growths on the valves. The particles are suddenly separated, and, being carried along with the stream of blood, become fixed in the artery. Recovery may ensue for a time; but (5.) Repeated subsequent attacks occur, each associated with palpitation and irregular action of the heart; and

with the cessation of the cardiac symptoms, sudden and simultaneous obstruction of peripheric arteries; (6.) At last gangrene ensues in some parts, and death results (MALMSTEN, of Sweden). In the brain such infarction leads to yellow softening. In the spleen and kidney the infarction consists of a conically-shaped mass of material exactly limited, of a colour varying with the size of the lesion, and generally more dense than the surrounding parenchyma (*Med. Times and Gazette*, vol. xvi., p. 273). The obstruction is generally at the point of narrowing of arterial branches immediately after bifurcation (*splenic pencilli*). (7.) Strong pulsation of vessel on cardiac side of occlusion (TUFNELL).

Venous Embolism occurs—(1.) When coagula arrive in the right side of the heart. Here they are attended with symptoms of exhaustion, pulse small and intermitting, followed by collapse and powerlessness of muscle. The patient, although inhaling deeply, seems to suffer from apnœa; the veins become highly turgid; and sudden death occurs if large concretions separate and suddenly obstruct the pulmonary artery; as in the case recorded by Druitt, and noticed at p. 668. The symptoms were,—hurried and anxious breathing; pulse rapid and scarcely perceptible; features intensely pale, bluish, and distressed; the whole surface of the body cold, but drenched in perspiration; no pain, but great agitation and feebleness; the air entered the air-cells freely, but the beating of the heart was a confused and feeble "*wobble*"—its rhythm and force were gone. The intellect was clear.

The diagnosis of embolism generally may be summed up as follows:—

I. *Phenomena of Pulmonary Arterial Embolism:* (A.) *Parenchymatous.*—(*a.*) Collapse of lung; (*b.*) Peripheral pleuritis of lung; (*c.*) Hæmorrhagic effusion; (*d.*) Capillary bronchitis, with cough and expectoration if the embolism is capillary. (B.) *Functional.*—(*e.*) A craving for air (anxietas): although a deep breath may be drawn without pain, yet every movement of the body tends to increase the anxietas; (*f.*) Lowering of temperature; (*g.*) Systolic murmur; (*h.*) Increased impulse of the right side of the heart; (*i.*) Irregularity of rhythm; (*k.*) Undulation of veins in the neck; (*l.*) Cyanosis, vertigo, albuminuria, œdema of limbs. These phenomena are seldom all present, death being too sudden.

II. *Phenomena of Systemic Arterial Embolism.*—These have

been already described on the previous page (Simpson, in *London and Edin. Monthly Med. Journal*, 1854, p. 281; *Med. Times*, Feb. 1, 1862, p. 112). The coagula, which exist without simultaneous lesions of the arterial wall, or adjoining capillary circulation, are never formed on the spot, but have been separated from some distant point in the circulation, and have been carried by the current of the blood as far as they could go. Such are genuine emboli. They are always found in places where a large arterial trunk, by giving off branches, suddenly acquires a more constricted calibre. Virchow, Paget, Malmsten, and Kirkes have shown independently of each other that—(1.) Fibrinous concretions may separate from the valves of the heart during life; (2.) That they may cause obstructions of particular peripheric vessels; (3.) That by admixture with blood they may have a poisonous decomposing influence similar to typhus or pyæmic poisons. In the lungs, for example, apoplectic foci become developed. Infarctions occur, and their results, in broad masses of fibrin, remain. On the arterial side of the circulation metastatic fibrinous wedges occur in the vessels; petechial spots occur on the skin and on mucous membrane, pericardium, and peritoneum. Similar fibrinous deposits accumulate in the spleen.

III. *Phenomena of Venous Embolism.*—The coagula found in the veins are the products of local stasis, often caused by roughness of their inner surface, by alteration of structure and relaxation of elementary parts. When the coagulum adheres only to one wall, with the effect of narrowing the passage, such a thrombus is never of an embolic nature. It is a clot, plug, or coagulum of fibrin formed or forming there as a result of local causes of irritation. (See previous description.)

IV. *Phenomena of Pigmental Embolism.*—A peculiar form of embolism is associated with accumulations of pigment in the blood. It is primarily developed in the spleen, whence the thrombi are conveyed to the *vena portæ*, gradually increasing in circumference. Malarious fevers establish such pigments in the blood (PLANER, of Vienna). In such cases sanguineous extravasations, in great numbers, are found in the brain; and abscesses in the liver: laceration of hyperæmic capillaries is also a result—*e. g.*, Malpighian glomeruli of kidneys.

From what has been written, it is therefore necessary to distinguish at least *three* forms of *metastatical dyscrasiæ*—namely,

(1.) *Metastasis associated with the phenomena of embolism, tending to the development of multiple centres of inflammation, and their suppuration;* (2.) *Metastasis in which there is something over and above embolism, and quite independent of its occurrence, and which may get well if secondary abscesses do not form in important viscera—septicæmia, ichoræmia* (S. RINGER); (3.) *Metastasis combining the phenomena of* (1.) and (2.)—*pyæmia.*

In the first class of cases death generally ensues from exhaustion; and when the multiple centres are in process of evolution, the febrile phenomena are always increased—a distinct attack, with rigor, being excited and associated with each centre of development. Evening exacerbations of fever are distinctly marked, so that the ranges of temperature during the course of the disease are similar to those of extensive suppuration attended with fever. The temperature, however, in some of these cases is not very high; but in both forms of metastasis mentioned the type of the fever is intermittent during the day (S. RINGER, BILROTH)—*i. e.*, the temperature between the hours of elevation becomes normal; and Professor S. Ringer writes that he has never met with any disease in which this occurs except *pyæmia.* It is no doubt due to this circumstance being hitherto overlooked that such varied statements are current regarding the heat of the skin in pyæmia. In some cases, indeed, the temperature is never very high, nor the intermittent elevations very numerous, yet these are the kind of cases in which the internal organs are apt to be studded with secondary abscesses.

In the second class of cases of *metastasis,* in which intense febrile phenomena occur, quite independent of embolism, the temperature is very high, the intermittent elevations are numerous, and frequently there are no multiple centres of inflammation in visceral parts (RINGER). Though the exact chemical change in the composition of the blood has not been made out, yet, as it has been produced experimentally by the injection of filtered putrid fluids into the veins or intestinal canal, it may be fairly concluded that such cases are due to the absorption of putrid poisons in a gaseous or other fluid form, especially resulting from the decomposition of tissues. Tissue-change or metamorphoses in the febrile state may also establish sources of blood-poisoning (*e. g.*, "Rheumatism"). Dr. Parkes states how the amount of urea rose in a case of pyæmia after amputation, from

3·1 for every lb. of body-weight to 4·26 grains. The weight of the body was 131 lbs. It fell to 110 lbs. in eleven days, and the temperature rose from 101° Fahr. to 105·6° (*On the Urine*, p. 178).

In surgical cases the phenomena commence to be expressed between the second and fourth day after an operation or injury. The tissue round the source of the mischief becomes red and œdematous towards the third day; and *ichor* may exude from the injured part, mixed with bubbles of gas. Disintegration spreads with great rapidity from the seat of injury; and where the part is to-day simply *œdematous*, to-morrow it will be an *ichorous* infiltration; and so it proceeds till death occurs. In some cases it may be looked upon as a progressive gangrene of areolar tissue (the acute purulent œdema of Perigoff). If thrombi form in the veins, and the phenomena of embolism occur, the danger is increased, and the case is more complex; but the *septic* phenomena, in the first instance, are quite independent of embolism. A very high temperature and a more continued fever are characteristic of septicæmia. The pulse rises (generally above 120), and the intense burning heat communicated by the skin of the patient is well known to the hand of the experienced surgeon. On the evening of the fourth day after an injury, Bilroth records the temperature at 37·3° C. = 99·5° Fahr. In the night the œdematous infiltration commenced, and on the ensuing morning the temperature was 40·1° C. = 105·8° Fahr. On the eighth day the patient died, up to which time the temperature remained high, its lowest point being 39° C. = 102·2° Fahr. A rapid and considerable increase of temperature on the *third* or *fourth* day, with delirium and a somnolent state, with an ichorous appearance of a wound, and œdematous infiltration around it, are certain signs of septicæmia. Albumen occurs in the urine in some cases, and sweats are common (Bilroth); the skin acquires a faint yellow colour; and the breath a hay-like smell. The highest temperature recorded by Professor S. Ringer is 107·4° (*MS. Notes*). Some of these cases undoubtedly get well, although a very large proportion die; and the chances of getting well are greater in proportion to the freedom from embolism, and from intense febrile disturbance as indicated by temperature, and from the formation of secondary abscesses. Dr. Gillespie and Mr. Spence, Professor of Surgery in the University of Edinburgh, have recorded undoubted instances of recovery from cases of this

kind; so also Mr. Prescott Hewett recounts ten recoveries within his experience.

Treatment.—The indications are—(1.) To remove all those conditions which favour *thrombosis* and the degeneration, metamorphoses, or putrefaction of blood-clots or tissue; (2.) To subdue the force of the circulating current, so as to prevent excitement during the long and slow metamorphosis of a clot when it has formed; (3.) To sustain the strength and allay the nervous irritability. Wine, brandy, rum, milk, strong animal soups, should be freely given from time to time, in small quantities. Opium should be given freely at night.

Free exposure of the patients to currents of fresh air—treatment in the open air, in fact—has been shown by Mr. Paget to be the most promising of success in cases of pyæmia,—by far the most fatal of all *traumatic infections*, which kills nearly all whom it attacks.

CHAPTER X.

DISEASES OF THE RESPIRATORY SYSTEM.

LUNG DISEASES.

The acute inflammatory diseases of the pulmonary apparatus are more or less distinctly defined according to the tissue which they implicate and the symptoms to which they give rise. The three structures which mainly take part in the constitution of the lung substance being—(1.) The *bronchial tubes*, terminating in (2.) The *pulmonary air-cells, vesicular structure, proper substance*, or *parenchyma of the lung;* and (3.) *The membrane covering this parenchymatous part*, forming a portion of that serous sac interposed between the lungs and the walls of the thorax. That portion of the serous sac which immediately invests the lungs is known by the name of the *pulmonary pleura*, while that which is applied against and invests the parietes of the thorax is known as the *parietal* or *costal pleura.*

One or more of those pulmonary structures may be associated in the processes and results of inflammation. Thus the bronchial membrane may be inflamed, when the disease is termed *bronchitis;*

or the substance of the lung may be inflamed, the disease being then called *pneumonia;* or the pleura may be inflamed—a condition which is described as *pleuritis.* It is rare, however, in practice to find that these morbid states are so completely isolated. More frequently, for instance, with *pneumonia,* or inflammation of the substance of the lung, there is associated more or less inflammation of the air-tubes (*bronchitis*), on the one hand, constituting *broncho-pneumonia;* or there co-exists inflammation of the investing pleura (*pleuritis*), constituting, on the other hand, *pleuro-pneumonia.* The physician can now distinguish each of these elementary conditions by definite symptoms; and by observing the combination of physical phenomena, their association with general symptoms, and the sequence of their occurrence, he is able to determine how far any given pneumonic affection involves one or more of the structures which compose the lung; and to direct the treatment of the case accordingly. It is therefore necessary to describe the phenomena of inflammation in each of these structures in detail.

PLEURISY.

Latin Eq., *Pleuritis;* French Eq., *Pleurésie;* German Eq., *Pleuritis*—Syn., *Rippenfellent-zündung;* Italian Eq., *Pleuritide.*

Definition.—*Inflammation of the serous membrane that lines the cavity and covers the viscera of the thorax. It is characterized at its outset by a febrile chill, followed by an acute sharp pain in some part of the chest, frequently called* "a stitch in the side," *as it is usually confined to one spot about the lateral regions of the thorax. The acts of respiration are performed rapidly, and are not completed. A dry short cough supervenes, and the pulse is hard and quick. The natural serous secretion of the pleural sac is arrested in the first instance, but soon becomes increased in quantity, and of an inflammatory type, the exudation tending to assume the* corpuscular *character. The effusion more or less rapidly increases, and may ultimately assume a sero-purulent character; and the parietes of the corresponding side of the chest may dilate accordingly.*

Pathology and Morbid Anatomy.—The inflammatory phenomena begin in the sub-pleural tissue, whose vessels enlarge and admit red blood, and shortly afterwards the red blood penetrates the web

of the pleura itself, and the process is more or less diffuse. At first a number of red dots may be visible, which at length are so multiplied as to become confluent and form large patches, which spread till perhaps the whole of the *pleura pulmonalis and costalis* is in one continuous state of inflammation. The membrane is in all cases of a bright red or arterial colour, slightly thickened from interstitial deposit, and easily detached, from the diminished cohesion of the sub-pleural fibrous tissue.

If the diffuse inflammation be of any intensity, the secretion from its surface is in general suspended at first, and the membrane is dry. In this state the inflammation may terminate by resolution, or serum may be poured out, forming the *serous inflammatory effusion.* The quantity of serum thus effused is extremely various. In some cases it hardly exceeds a very few ounces, while in other instances it amounts to many pints, separating the usually opposed surfaces of the membrane, and distending the cavity of the pleura, and compressing the lung. Laennec is of opinion that the time of effusion after the commencement of the inflammation is often very short, as he has detected ægophony and absence of respiration, as well as of thoracic resonance, an hour after the patient has first felt pain in the side. If the effusion be considerable, the lung becomes collapsed, contains no air, and therefore no longer crepitates; the vessels are devoid of blood, while the bronchii, even to the large trunks, are evidently contracted; still, if this lung be inflated, it enlarges more or less perfectly. Again, should the pleuritic effusion be less in quantity, some fluid appears spread all over the lung; but the greater quantity is collected at the lower portions of the chest.

Accompanying either of the previous forms, or existing *per se*, the *fibrinous inflammatory lymph* may predominate, and adhesion of the opposed surfaces ensue. In many cases the lymph is loose and watery, rendering the serum turbid or flocculent; but in other cases it is more solid, and adheres with great tenacity to the opposite membrane, becoming organized at both surfaces. The organization of these membranes is rapid, and is often effected in the course of forty-eight or even twenty-four hours. If the patient dies shortly after an attack of acute inflammation, these adhesions are found soft, easily lacerable, and extensible. If, however, he survives a longer period, the adhesions are often of

great tenacity, are indurated, and with difficulty separated from their attachments. The extent of membrane affected with adhesions is sometimes limited to a small portion, and sometimes extends over the whole surfaces of the cavity; but their most common seat is over the anterior lobes of the lungs, or the portion of pleura from the mamma to the axilla.

Empyema.—The pleuritic inflammation sometimes terminates in suppuration; and should the pus be in such quantity as to accumulate in the cavity of the chest, the disease is termed *empyema*. Empyema may be true or false; it is said to be true when the pus is secreted by the pleura, and false when it results from the bursting of an abscess of the lung into the cavity of the chest. The quality of the pus in true empyema varies from a genuine laudable pus to a sero-purulent fluid. In quantity, also, it varies from a few ounces to many quarts, filling the entire cavity of the chest. Under these latter circumstances the side of the chest is dilated, and the intercostal spaces are widely separated and bulging.

Effusion of pus may take place into either cavity of the chest, but the left, perhaps, is the more common. The phenomena accompanying empyema of the left side are remarkable; for, besides the lung being found collapsed, and not so big as the fist, the heart is sometimes seen transposed as far over on the left side as it usually is on the right. In cases, however, in which *paracentesis* has been performed, and the pus has been drawn off, the heart is observed to return to its place, while the lung, less completely collapsed, may be bound down to the upper and lower portion of the chest by long and multiple adhesions.

General Symptoms of Pleurisy.—Like other inflammations of the lungs, pleurisy may be acute or chronic. The acute form of this disease may be preceded by fever, but often no such antecedent is present. Its local symptoms, however, in most cases are strongly marked, the patient suffering with severe *continued pain* in the affected side, of "a dragging, shooting character," which is greatly exasperated by coughing or forced inspirations, movement, pressure, and percussion, so that the lungs can only be imperfectly filled with air. The seat of the pain, however extensive the inflammation, is generally limited to one point; and this point is usually about the centre of the mamma, or just below that part, towards the lateral attachments of the diaphragm. While the pain is constant, it nevertheless sometimes remits, and with

the occurrence of effusion often totally disappears. The tongue is commonly white, but the pulse varies, perhaps according to the form of the inflammation and its intensity. If the disease be limited to an effusion of lymph or serum, the pulse is seldom more than 90 to 110, but "hard and concentrated in impulse." Either form of *pleurisy* is generally accompanied by a short, troublesome cough and some expectoration. The respirations are increased in frequency—phenomena of more constant occurrence than even the local pain—and, unless dyspnœa exist, are unnoticed by the patient. While one *respiration* is performed, only *three* beats of the heart, in place of *five* or *six*, occur. The patient likewise is for the most part restless, and lies on the affected side; but if the effusion is great, the lung is compressed, which increases the general uneasiness, as well as the oppression of the breathing; and the patient, instead of lying on his side, now lies on his back, or sits propped up in bed. If he recovers, the fluid effused is absorbed with greater or less rapidity, and his amendment is proportionally retarded or accelerated. In fatal cases, although the lung may for a time become accustomed to the altered state of things in the chest, yet fresh effusions occur, which shortly terminate the life of the patient.

The duration of the disease is such that acute pleurisy sometimes terminates in a few hours, sometimes in a week or ten days, while cases have been met with in which many months have elapsed before the pleuritic effusion has been absorbed and the patient restored to health.

Symptoms of Empyema.—Again, if the inflammation is about to issue in the formation of pus, the pulse is extremely small and frequent (from 120 to 150), while the restlessness and anxiety of the patient are greatly increased. There are cases, however, of empyema in which the patient suffers little pain, or any more marked symptom than usually awaits the last stages of phthisis. In some instances he is for a time even capable of walking about the ward of an hospital or in a bed-room. Supposing, however, empyema to have occurred, any acute pain which may have existed subsides, but the anxiety of the patient is increased, and his state of collapse shows his imminent danger. If the constitution be less affected, the symptoms vary according to the side of the chest which is the seat of the empyema. If it be on the left side, for example, the heart is often transposed, and felt beating as far over on the

right side as it usually does on the left, and the pulse is small and frequent. If we now bare the chest of the patient, we find the affected side enlarged, sometimes œdematous, with projecting intercostal spaces. As the lung is now greatly compressed, no respiratory action is seen on that side, which is entirely at rest. If *paracentesis* be now performed, the heart is restored to its place as the pus flows; but as the lung for the most part only imperfectly expands, the affected side, even in the most favourable cases, contracts, and the spinal column, pressed upon by an unequal weight, acquires a lateral curvature, the shoulder sinks, and the patient is greatly and permanently deformed.

Auscultation and percussion are equally valuable in determining the amount of effusion. If serum or pus be effused to the amount of a pint, for instance, the lung is displaced to that extent; and consequently the lower portion of the chest, when struck, returns a dull sound, which extends as high as the level of the fluid. If we now auscultate the patient, the respiration is also lost below the level of the fluid. Besides these results, the voice gives very striking indications of the lung becoming so far condensed from the pressure of the fluids; for we very constantly have *bronchophony*, and occasionally *ægophony*.

If the chest be completely filled in empyema, the respiratory sound is altogether wanting; so is *ægophony* and *bronchophony*, and the containing cavity returns a dull sound at whatever part percussed. Under these circumstances, and especially if the heart be displaced, the affected side will be seen entirely motionless, rounded, and distended; and when these signs are present, there can be no doubt that the distension is due to the presence of serous effusion or pus. But the physical signs of pleurisy are much more delicately varied according to the anatomical conditions of the disease. For example, at the most early stage, when the serous secretion is at first arrested, the expansion of the walls of the chest is diminished, as may be proved by measurement. The percussion-sound, however, is not perceptibly altered, and respiration is weak, because imperfectly performed. The characteristic *friction-sound* of inflamed serous membranes may, perhaps, now be detected, if listened for in the *infra-mammary* or *infra-axillary* regions. When the secretion has returned, increased in quantity, the signs continue as described, but the clearness of the percussion-sound becomes diminished, and the

friction-sound is of a rubbing or grating character. The period of inflammatory effusion is now established, and the infra-mammary and infra-axillary regions become more or less bulging; the projection of the intercostal spaces of the affected side during both respiratory acts becomes most obvious; the thoracic vibration from the voice is abolished where the fluid intervenes, and so also are friction-murmurs there. The area of dulness, and of the peculiar sounds, may be changed by altering the position of the patient. The natural respiratory murmurs become greatly intensified above the level of the effusion. When the effusion exists on the right side, the sounds of the heart are more clearly audible than in the natural state in the right axillary region, because the lung is more solidified by the pressure of the effused fluid.

Such are the more salient symptoms which mark the progress of pleurisy. The disease, however, may exist without any of the general symptoms. There may be neither local pain, cough, dyspnœa, nor febrile action, and yet effusion may have occurred to such an extent as to have reached the clavicle, while the patient remains utterly unaware that his chest was the seat of disease (WALSHE). The physical signs alone reveal the disorder—which is termed *latent pleurisy*—a form of disease which had no existence in nosology previous to the time of Laennec. Whenever, therefore, the least suspicion exists of disease in the chest, especially in elderly persons, or those liable to constitutional affections, percussion and auscultation must never be neglected.

Causes.—The causes of pleurisy have an influence over its course; therefore a due appreciation of them is essential to a correct prognosis and treatment. The inflammation is sometimes said to acknowledge an idiopathic origin, as when the pleurisy is believed to arise from exposure to cold; but it usually results during the progress of some *specific* or *constitutional* disease. Exposure to cold, especially to currents of air, when the person is heated, is a frequent exciting cause; but many now express their entire disbelief in cold being able to establish pleurisy in a healthy person (HYDE SALTER, FULLER). When exposure to cold is followed by inflammation of the pleura, the disease is most likely associated with some morbid condition of the blood; and without doubt blood-poisoning is one of the commonest morbific causes of serous inflammations. For example, *anæmia*, *pyæmia*, the specific

toxæmia of *eruptive fevers*, the *materies morbi* of *rheumatism, gout, scrofula* (*tubercular pleurisy*), *carcinoma, Bright's disease, alcoholism, syphilis, or even retained excreta, with pyrexia as the result of over feeding*, are all apt to set up serous inflammation. All serous inflammations, in truth, especially point to states of blood-poisoning; as during the progress of typhus or puerperal fever, or during Bright's disease, or the pleurisy may acknowledge some adjacent irritation, as *pneumonia*, constituting *pleuro-pneumonia*, or the irritation of some growth in the lungs, such as *tubercle* or *cancer*.

Prognosis.—Simple idiopathic pleurisy on one side of the chest, occurring in a person whose lungs are not chronically diseased, almost always terminates favourably, if taken in time and treated judiciously and with energy, and if the effusion has not been copious; but when it occurs as a complication in other diseases, the result may be doubtful, especially if air finds its way into the cavity (*pneumo-thorax*).

Treatment of Pleurisy.—In acute pleurisy, during the first stage, or that of *hyperæmia*, the best practitioners of all times and of all countries have taken blood from the arm, provided the strength be good and the symptoms sthenic; and if, says Laennec, after one or two bleedings, the pain in the side and fever have not abated, blood should be taken from the side by leeches or by cupping. The practitioner should also remember that effusion often takes place after bleeding, during the subsidence of the inflammation, so that the breathing is often more oppressed, and the symptoms for a time aggravated, although the condition of the patient is in reality improved. The lung, however, soon gets accustomed to this new state of things; and the fluid in a few hours beginning to be absorbed, the symptoms are generally ameliorated. Hot poultices and leeches should be always employed when pain on inspiration is present, of a "catching" or "stabbing" nature. *Tartar emetic*, says Laennec, is in general well supported in pleurisy, and contributes powerfully to subdue the inflammatory tendency; but, nevertheless, when the pain in the side and fever have ceased, it loses further power over the disease; at least, it does not appear to promote the removal of the fluid effused, so that its use must generally be abandoned as soon as the acute symptoms have passed away.

With respect to the application of blisters, Laennec objects to

their use until the acute stage is past; but when the pain has ceased for some days, and absorption of the fluid proceeds slowly, and the disease promises to become chronic, a succession of blisters may be applied.

Such is an outline of the treatment recommended by Laennec, and which is still to be followed out. The points aimed at by venesection being relief of pain, and moderation in the force and frequency of the pulse, the patient ought to be bled in the upright posture, and the blood should be allowed to flow in a full stream until he can take a deep breath freely, or till he feels faint and exhausted. From ten to twenty ounces may be necessary to accomplish this end, according to the severity of the case and the nature of the constitution. After free evacuation of the bowels has been effected, *calomel*, to the extent of producing the *slightest* mercurialization, is the most beneficial line of treatment, to be followed *in certain cases only*—namely, those which do not acknowledge any *constitutional disease* as their cause. The more rapidly slight mercurialization can be produced the better; and hence, writes Dr. Walshe, during the first six hours, small doses of *calomel with opium* (*a grain and a half of the former*, combined with *a sixth of a grain of the latter*, or more, if the pain continues acute) should be given every half hour, while mercurial ointment is rubbed into the skin of the affected side near the axilla every fourth hour. Dr. Fuller recommends *half grain* or *grain doses* of *opium* every *three* or *four* hours, in combination with *one* or *two grains of calomel, and half a grain of digitalis*, and to have *the whole side covered with a piece of linen spread with mercurial ointment:* over this is placed a poultice covered with oiled silk (l. c., p. 184). The patient must be carefully watched, so that neither *ptyalism* nor *narcotism* is produced. The moment mercurial action has been established, the further administration of the mineral must cease.

Dr. Bennett has not met with a single instance where the good effects of calomel have been unequivocal (*op. cit.*, p. 654).

Opium and digitalis are advised to be continued after the use of calomel has been suspended; and with them may be given two grains of *squills* or of nitrate of potash, which will act beneficially as a diuretic; or after twelve or more hours very small quantities of *tartar emetic* in solution may be given at night, combined with small doses of *opium* and *ipecacuanha*, to allay cough and

general irritation. After the febrile action has in some measure subsided, and the active stage of the disease is at an end, a blister may be applied over the lateral region of the chest, but *not* over the seat of pain; and if the fluid continues to accumulate, the blisters ought to be repeated, so as to maintain a surface at a distance from the affected part in a constant state of counter-irritation. Diuretics ought at the same time to be freely given. The *compound tincture of iodine*, in doses of *twenty minims freely diluted*, is a valuable medicine at this juncture; and so also is the *liquor iodum* of the British Pharmacopœa of 1867.

In the chronic stage of pleurisy, physicians are agreed as to the necessity of generous diet and tonic remedies in aid of any diuretics and absorbents which may be employed. If the patient be kept too low after the stage of active inflammation has subsided, or be unduly depressed, it will not only be impossible to induce absorption of the fluid, but there will be great danger of its becoming sero-purulent in character. The unfavourable issue of pleurisy in its chronic stage is in many cases attributable to a want of tone in the system, caused by the treatment adopted; the patient being kept too low, or he is overmuch purged, or is in some other way unduly depressed, so that the system is unable to exercise its reparative power. A more generous diet ought to be given, and the general health sustained by quinine and other tonics. The use of diuretics and absorbents ought, at the same time, to be steadily persevered in. When a succession of blisters has failed in relieving the patient, *iodureted lotions*, or *iodureted ointments*, combined with the internal administration of *cinchona* with *tincture of iodine*, *iodide of potassium*, and small doses of *bichloride of mercury*, *nitre*, *acetate of potash*, *squills*, *digitalis*, *and cantharides*, have each in their turn effected the desired object (l. c., p. 186). The fluid has been gradually re-absorbed, and recovery has ensued. The formulæ most useful in such cases are—(1.) A diuretic in the form of a pill composed of *digitalis*, *squills*, and the mass of *pill hydrargyri*, of each a grain and a half; *nitrate of potash* (twenty grains), combined with *tincture of the perchloride of iron* (fifteen minims), three times a day, may at the same time be given (Fuller, Chambers). Professor W. T. Gairdner recommends as a diuretic the *cream of tartar* electuary, in which the *cream of tartar* is mixed in equal proportions with treacle, honey, or

marmalade, and in some cases flavoured with a few drops of peppermint oil. The dose is a teaspoonful repeated as often as the stomach will bear it, or as the urgency of the case demands.

(2.) A lotion, to be applied over the chest by *spongeopiline*, or by *lint* covered with *oiled silk*, composed as follows:—

℞. Hyd. Bichloridi, gr. iv.; Tinct. Iodinii Co., ʒiv.—ʒvi.; Glycerini, ʒiii.; Aquæ Distillatæ, ʒivss.; Ft. Lotio.

(3.) One or other of the following ointments may also be rubbed in upon the skin, over the side of the chest: namely—

℞. Hyd. Bichloridi, gr. iv.—v.; Ungt. Iodinii Co., ʒiv.—ʒvi.; Adipis, ʒiv.—ʒi.; Ft. Ung.; or—

℞. Hyd. Bichloridi, gr. iv.—v.; Potassii Iodidi, ʒii.; Aquæ Distillatæ, q. s. ut Hyd. Bichlorid. et Potassii Iod. solventur; Adipis, ʒi.; Ft. Ung.

Professor W. T. Gairdner and Dr. Fuller are the two most recent authorities who notice the treatment of pleurisy by puncturing the chest and letting out the fluid—an operation to which the name of *paracentesis* has been given. With regard to acute pleurisy, Dr. Gairdner infers, from his own experience, that "for the mere saving of life in the acute stage of the disease, the operation is not necessary in any but a very small minority of the cases which have not yielded to remedies" (*Clinical Medicine*, p. 374). But a large proportion of the cases recorded in Dr. Gairdner's excellent work, just quoted, occurred before he began to adopt the method of withdrawing the fluid practised so successfully by Dr. Bowditch, an American physician. In consequence of the great facilities afforded by this method for withdrawing fluid from the chest, Dr. Gairdner's views regarding the operation have undergone considerable qualification. He now substantially gives in his adhesion to the principles and practices of Dr. Bowditch, and claims for therapeutics a correspondingly extended field of usefulness. He has only performed the operation in acute pleurisy in cases of *great distension*, after other remedies had a fair trial without effect; and has avoided doing it in cases of partial or moderate effusion.

As a means of relieving the chest in chronic pleurisy and empyema, the operation is a useful one; and the practical question is as to the time when the operation should be performed. The

condition of the patient's health and respiration, and the absorption or non-absorption of the effused fluid, are the aids to a decision for or against the performance of the operation. As long, writes Dr. Fuller, as the breathing is not seriously embarrassed, and the general health does not decline, so long are we justified in making full trial of our remedies. But as soon as extreme shortness and distress of breathing, or lividity and anxiety of the countenance, denotes serious interference with the functions of life, delay is no longer justifiable, and it becomes our duty at once to give our patient the chance which the operation affords.

The space between the fifth and sixth ribs, counting from above downwards, should be selected (if adhesions are ascertained not to exist there), being the most depending part of the chest when the patient lies on his left side, the more usual position in this disease.

The operation is described under the following subject, namely:—

HYDROTHORAX.

LATIN EQ., *Hydrothorax;* FRENCH EQ., *Hydrothorax;* GERMAN EQ., *Hydrothorax*—Syn., *Brustwassersucht;* ITALIAN EQ., *Idrotorace.*

Definition.—*Watery fluid or serum in the cavity of the pleura (of either or both sides), rarely occurring in the absence of pre-existing disease of the pleuræ, lungs, heart, or great vessels, nor without the influence of some specific or constitutional disease—"passive dropsy of the pleura."*

Pathology.—In hydrothorax the cavity of the chest, on being opened after death, is found more or less full of watery fluid or serum, which being removed, the pleura is seen sometimes healthy, but more generally of a dark colour, in consequence of a quantity of venous blood being congested in the vessels. The fluid may be effused into one or into both cavities. It may also be limpid and colourless, like water; but more commonly it is citron-coloured, and contains fibrin, much albumen, and sometimes urea, in cases of *Bright's disease.* The quantity effused varies from a few ounces to many pints: eight and nine pints are not unusual; and Laennec states that he once removed twelve pints from the right side of the pleural cavity. When the quantity of fluid is large,

the lung is compressed towards its roots, and placed generally in the groove formed between the sides of the bodies of the vertebræ and the heads of the ribs to their angles. It is sometimes so flat as not to be more than half an inch in thickness. It may be compressed against the sternum if previously-existing adhesions to that region have fixed its position there. When hydrothorax is secondary, almost every chronic affection, either of the liver, kidney, or heart, may be found co-existing at the same time. Occasionally it is the result of extremely slight pleuritis, rarely of severe pleurisy, and in these cases the serum is more flocculent, contains more albumen, and portions of lymph are often also seen adherent to the *pleura pulmonalis* or *pleura costalis;* the two pleuræ are also often more or less united.

Symptoms.—The effusion may take place either gradually or suddenly. In the former case it may be so slow that the lung is able to adapt itself to the presence of the accumulating fluid, and the symptoms will consequently be much less marked, although the effusion be large. In the latter case the functions of the lung are almost at once suspended, the countenance livid, and the breathing greatly disturbed. When the effusion is slow, the symptoms are,—difficulty of respiration, which is carried on rather by the shoulders and diaphragm than by the intercostal muscles, some expectoration, lividity of the face or lip, œdema of the legs, and either a very full labouring pulse, or one that is small, frequent, and intermitting: the urine also is extremely scanty. As long as the effusion is moderate the patient can lie flat in his bed without experiencing any inconvenience. In the event, however, of the effusion being so considerable that the function of the lung is entirely suspended, the patient is unable to lie down, from the sense of suffocation produced by the fluid gravitating towards the root of the lung, and compressing the larger bronchi, and he therefore sits propped up by pillows, with his head bent forwards. When hydrothorax is symptomatic, or consecutive of affection of the heart or of other disease, it is generally preceded by swelling of the legs or eyelids, by the urine being plentiful and albuminous, or scanty, high-coloured, and loaded with the usual salts, and indeed by most of the symptoms of dropsy generally. In these cases the effusion seldom takes place into the chest till a few days before death, rendering the agony of death doubly painful and suffocating.

When the effusion is moderate, auscultation gives bronchial respiration, some mucous rhonchus, and *bronchophony*, and occasionally that condition called *ægophony*, which is a broken sound like the bleating of a goat, or the notes used in the exhibition of "Punch," and which is heard as though the patient was speaking at the end of the stethoscope, but not through it. This singular phenomenon is heard only when the instrument is placed about the level of the effused fluid. When the effusion is more considerable, the respiration is almost tracheal, there is neither *bronchophony* nor *ægophony*, and a dull sound is returned over a greater part of the chest. Again, if the patient's chest be bared, there is no expansion on the side of the seat of the effusion, the respiration of that part being carried on altogether by the shoulders and diaphragm; and should the effusion be excessive, the affected side bulges out, as in empyema, and its intercostal spaces are enlarged and prominent. If air exists as well as fluid, succussion of the patient gives the sound of the splashing of fluid, and sometimes the patient can produce this phenomenon by shaking his body himself.

Diagnosis.—The absence of pain and of the other symptoms of inflammation distinguishes this disease from *acute pleurisy*. Should, however, the pleurisy be chronic, it is impossible to distinguish the two diseases except by the previous history. The diagnosis, also, between *hydrothorax* and *œdema of the lung* is not always easy.

Prognosis.—Some cases of hydrothorax recover, but the prognosis is in all cases extremely grave and doubtful.

Treatment.—The treatment of hydrothorax is of great difficulty, from the many causes on which the effusion may depend, and also from the almost uniformly intractable nature of the disease. The general principles of the medicinal treatment to promote absorption of the fluid have been given under the section on the treatment of pleurisy; and it only remains here to notice the great facilities afforded for the removal of the fluid by the operation recommended by Dr. Bowditch, with the apparatus devised by Dr. Morrill Wyman, of Cambridge, U.S. The apparatus consists of a trocar a little larger than the ordinary exploring trocar, and a silver canula, with a stop-cock in silver, as light and small as possible, capable of being connected with a syringe by an intermediate piece of brass, also provided with a stop-cock, the two cocks working the same way, and acting as checks upon

each other. Such instruments may be had of Mr. Kemp, Philosophical Instrument Maker, Infirmary Street, Edinburgh; and a drawing of the instrument may be seen in vol. xxiii., p. 348, of the *American Journal of Medical Science;* also for Jan., 1863; also vol. xx., Oct., 1850.

During the operation the patient should be seated, when possible, sideways on a chair, or astride the chair, with his face to the back of it; or, if unable to rise, he ought to be brought so that the affected side may be made to incline slightly over the edge of the bed. The most appropriate spot for puncture is between the seventh and eighth, or the eighth and ninth, or the ninth and tenth ribs, in a line let fall from the lower angle of the scapula; but as a rule let the trocar be introduced as low down as possible, consistently with the safety of important organs in the chest or abdomen. The exact position of the liver and spleen must be determined first in every instance. Laennec himself once transfixed the diaphragm and pierced the liver, and that through the *fifth* intercostal space. An enlarged liver or spleen may be detected as high as the fifth rib. Dr. Watson once witnessed an operation in which the trocar was pushed through the diaphragm into the spleen, which was unusually large. The patient died a day or two afterwards of peritonitis. Just under the lower angle of the scapula is a spot easily reached, and where the muscles are thin. But in selecting the precise intercostal space, Dr. Bowditch chooses one about an *inch and a half higher* than the line on a level with the lowest point at which the respiratory murmur can be heard in the healthy lung of the opposite pleural cavity. Having pressed the forefinger of the left hand deeply into the intercostal space, the trocar, with its canula, should be plunged through the tissues at the depressed part, keeping as near as possible to the upper edge of the lower of the two ribs. The point of the instrument should be raised rather than depressed, so as to avoid injury to the diaphragm, liver, or spleen. The skin need not be incised before puncture. Having withdrawn the trocar, leaving the canula, the double-valve syringe is to be applied, and the effusion slowly drawn away, until the lung has undergone as much expansion as it can endure with safety. This will be indicated by a sense of dragging distension or pain. When the canula is removed, the wound contracts and closes so completely that no lint or dressing is required.

In the hands of the American physician, Dr. Bowditch, and Professor W. T. Gairdner, of Glasgow, and Dr. Budd, of London, this operation has been the means of saving many lives. It is comparatively harmless, gives but little pain, and in the opinion of these eminent physicians it is an operation which ought never to be allowed to fall into disuse by the profession. The effect of the operation is to relieve the mind as well as the lung of the patient from great oppression. Although before the operation he is quite weak, he is often able after it to get up and walk. Digestion becomes at once improved, and strength is rapidly regained. The cough, however, is apt to augment during the first few days; the pulse also retains its quickness; friction-sounds occasionally become developed; and several months may elapse before the vesicular murmur becomes properly re-established in the lung.

Whenever the pleural cavity has become distended with fluid, and the dyspnœa is great, Dr. Bowditch recommends operation without delay. When thus performed early, it prevents a long and tedious illness, and future contraction of the chest. It should also be resorted to in all chronic cases when the effusion does not disappear after a reasonable time and the use of appropriate remedies. Dr. Bowditch has performed the operation 150 times on 75 persons, and has seen it done in 10 other cases. Out of the 75 cases, 29 recovered completely, and apparently in consequence of the operation, which was generally performed after severe symptoms had set in; and in all these cases the tapping seemed to be the first step towards recovery. In 26 of the 75 cases the fluid obtained at the first tapping was *serum;* and 21 of these cases made good recoveries. If the fluid afterwards became *purulent,* an almost certain fatality attended such a change. Of 6 such cases, 4 died; and the two others were likely to die when Dr. Bowditch wrote. In 24 of the 75 cases, *pus* flowed at the first tapping; and 7 of these recovered, and 7 died. In such cases relief is always obtained; but the tendency remains to a fistulous opening or to phthisis. If the fluid at the first tapping is sanguinolent, thin, and of a dark red colour, not coagulating, it forebodes an almost certainly fatal result; and is generally associated with some malignant disease of the lung or pleura. Of 7 such cases, 6 died; and the seventh was still lingering when Dr. Bowditch wrote. A mixture of bloody purulent fluid at the first

operation is usually fatal. A fetid gangrenous fluid is very rare, is obviously of bad omen, and betokens gangrene of the pleura and lung.

The operation may require to be repeated. Dr. Bowditch has done it *eight times* in six weeks to the same patient—himself a physician. One lady he tapped *nine times* in eight months and a half, commencing when she was four months and a half pregnant, and when orthopnœa was threatening death. She was delivered of a living child at the full time, and recovered. In one remarkable case a youth had obscure symptoms for nine months. Dr. Bowditch recognized by the physical signs "latent pleurisy," or "idiopathic hydrothorax," as some may style it. Four pints of fluid were removed at one time, and the lung was fully expanded in forty-eight hours. In three weeks the patient was well, and he continued so.

Dr. Bowditch, in a letter to Dr. Gairdner, thus states the general results to which he has arrived in the use of this operation (*Clinical Medicine*, p. 720):—"I now never operate unless I find some distension or rounding out of the chest, and filling up of some of the intercostal spaces, so that the chest presents a uniform curve, and not alternate depressions and elevations, as in the healthy chest. I operate under the following circumstances when I feel certain there is fluid:—

"1. When there is *severe permanent dyspnœa—orthopnœa*—however acute the disease, if I find fluid filling the pleural cavity, or nearly filling it.

"2. When there are occasional attacks of *orthopnœa* threatening death, even if there be not sufficient to fill more than half of the cavity. If the fluid seems to be the cause of the dyspnœa, I operate, because occasionally I have lost a patient when waiting for more extensive physical signs. This rule I apply to acute and chronic cases.

"3. I use the trocar after three or four weeks of ineffectual treatment, without any absorption being produced.

"4. In chronic idiopathic hydrothorax, a latent pleurisy, with simply physical signs to indicate *extensive* effusion, but when the rational signs are either very slight or none at all, save a general malaise and weakness."

In the experience of Dr. Watson the operation of *paracentesis thoracis* in simple pleurisy is not to be performed unless the life

of the patient is in jeopardy—that is, in cases "in which the effusion continues and increases, and the side, instead of shrinking, enlarges; the functions of the lung on that side are entirely abolished; nay, the use of the remaining lung is greatly interfered with by the pushing over of the mediastinum; and the patient is in imminent danger of suffocation." In such cases the oppressed lung must be relieved by "*letting the fluid out*" (Watson's *Lectures*, 4th edit., vol. ii., p. 128). Again, he writes, "Life is plainly in jeopardy when the vital functions of the lungs or of the heart are greatly hindered; when symptoms present themselves of approaching death by *apnœa* or by *syncope*. If we discover no cause for those symptoms except the increasing pressure of liquid pent up in the pleura, we are warranted in ascribing them to such pressure, and bound to act upon that persuasion." Also, if death by *asthenia* appears inevitable, the patient losing ground from day to day, and when all other means of getting rid of the pent up fluid have failed, the patient should not be denied the *chance* which the operation affords. Also, "whenever the effused liquid consists of *pus*, it should be let out" (l. c., p. 130). With the arrangements of Dr. Bowditch's syringe, we are enabled to remove fluid *before* the *false membrane* thickens over the compressed lung, and so makes it difficult to expand again. The trocar and small canula take the place of an exploring needle, which Dr. Watson does not object to use, regarding its use as a "minor diagnostic puncture;" and if the lung is not covered by an inexpansible *false membrane*, it will gradually expand as the fluid is withdrawn, the operation being done with the greatest slowness possible.

By the peculiar construction of the exhausting syringe, Dr. Bowditch has been enabled to evacuate the chest much more completely than by any other method, and to prevent entirely (and with absolute certainty) the admission of air. "It appears to me," says Dr. Gairdner, "to be in every respect an improvement so important that it may be said to open up a new history for the operation of *paracentesis thoracis;* and I trust it will receive in this country the attention which is due to it" (*Clinical Medicine*, p. 380). On the other hand, and by Dr. Bowditch's own showing, it does not appear that the entrance of air into the cavity of the pleura produces dangerous symptoms. He has never found this to be the case, even

when air has been pumped into the chest. Why, therefore, resort to so elaborate an arrangement of stop-cocks, syringes, and canulæ, and tubes? Dr. Fuller regards such instruments as practically inoperative. "The lung," he says, "is seldom able to expand freely at once; and as the chest walls will not yield beyond a certain point, air must be allowed to find ingress into the pleural cavity, or the fluid would not flow out. I have seen two of the most perfect of these stop-cock instruments employed; and so long as precautions were taken to exclude air from the pleura, so long they failed to draw off more than a few ounces of the fluid: directly air was admitted, the liquid flowed through them freely. Therefore, however desirable it may be theoretically to exclude air from the pleura, it is practically impossible to do so if we wish to relieve our patient" (FULLER, l. c., p. 192). But Dr. Fuller agrees in the view that "when acute inflammatory action has subsided, the admission of air does not necessarily excite suppurative action of the pleura,—nor is it found to interfere with the re-expansion of the lung. He believes that the lung is very slow in regaining its due expansion—more than five days. Yet he agrees also that every precaution should be taken to prevent admission of air (as it is certainly conducive to suppurative inflammation) when the grooved needle has shown that the effused liquid is serous (l. c., p. 193). Hence, I think, we come back to Bowditch's plan as the best. "When, on the contrary, the grooved needle has proved the existence of pus," Dr. Fuller believes "that the admission of air is not of the slightest importance." But here, again, we know that pus formed internally and not in contact with air, undergoes changes when exposed to air, which renders it not less imperative that we should, if possible, prevent its admission; therefore, if the lungs are not bound down by adhesion, or prevented from expanding by the thickness of the *false membrane* which covers it, here again Bowditch's syringe and stop-cocks enable the fluid, even when purulent, to be withdrawn without air being admitted.

But when air has been admitted, and when the fluid within the cavity of the chest has become changed thereby—is purulent and perhaps fetid—the evacuation of the contents by "drainage" seems the most efficient remedy. This operation consists of introducing, through the opening made in the chest for removing the fluid, "a fine, long iron probe, somewhat bent." It "is then

directed towards the lower and back part of the pleural cavity,—the lower the better. If the end of the probe be made to press against the side of the thoracic walls, it can be felt from the outside, through the intercostal spaces, though perhaps obscurely, owing to thickness and toughness of the false membrane. The lowest and most appropriate site in which the probe can be felt having been selected, an incision is made upon the end of the probe, which is then brought through the opening thus made. A strong piece of silk thread is passed into the eye of the probe, and drawn through the two openings, and the drainage tube—an india-rubber tube, perforated at frequent intervals, in the way recommended by Chassaignac for the treating of sinuses—being firmly tied to one end, is then drawn through by means of the silk: the ends of the tube are then tied together, and the operation is complete. . . . The openings in the chest walls are thus always free; the matter is discharged drop by drop as it forms, so that if the tube be suitably placed, there is never any collection of pus in the thorax; no time is given for decomposition, and the pus, therefore, is discharged in a healthy and pure state," (Goodfellow and De Morgan, in *Med.-Chir. Trans.*, vol. xlii.) These are just the conditions required in cases of empyema; and by means of such a tube fetid fluid may be got rid of, and the cavity of the pleura washed out with warm water, containing a weak solution of *permanganate* of potash (Condy's fluid), of the strength of *two fluid drachms* to the pint of water (FULLER). Dr. Watson is also of opinion that if the corrupt and corrupting mass, in two cases which he cites, had been duly removed, the patients would have had a much better chance of life. With such a tube and a syringe, offensive gases and fluids may be got rid of.

PNEUMOTHORAX.

LATIN EQ., *Pneumothorax;* FRENCH EQ., *Pneumothorax;* GERMAN EQ., *Pneumothorax;* ITALIAN EQ., *Pneumotorace.*

Definition.—*A collection of air or gas in the cavity of the pleura, generally co-existing with fluid in the same cavity,—when the condition is sometimes termed* hydro-pneumothorax.

Pathology.—Such a collection of gas or air within the cavity of the pleura, on either or both sides, occurs under any of the following conditions (FULLER, l. c., p. 199):—

1. When no communication exists between the pleura and the external air, it is then presumed to be due either to the spontaneous evolution of gas from decomposing fluid in the pleura, from gangrene of the pleural membrane, or from the secretion, generation, or exhalation of air from the pleura—a phenomenon of doubtful occurrence *per se.*

2. When a communication takes place between the pleura and the alimentary canal, as in cases of softening and perforation of the œsophagus or stomach.

3. When a communication occurs between the pleura and the atmosphere, through an opening in the chest-wall, the result of penetrating wounds of the thorax or of parietal abscess.

4. When a communication occurs between the pleura and the bronchi; either the *result of violence,* rupturing the lung-substance, and tearing the pulmonary pleura; or the *result of disease,* causing perforation of the pulmonary pleura from without inwards, as in cases of *empyema;* or from within outwards, by ulceration. Such ulceration may be due to *tuberculous disease,* to *hydatid cysts, cancerous growths, pneumonic gangrene, metastatic abscess* or *bronchial gland abscess;* or, lastly, the lesion may be the consequence of *emphysema,* or *pulmonary apoplexy,* or minute bronchial abscesses perforating the lung during the course of typhoid fever (GAIRDNER).

Numerous cases of pneumothorax are to be met with from the bursting of a pulmonary cavity—during inspiration; or from the opening of a pleural effusion into the lung. Indeed, tubercular ulceration of the pulmonary pleura is the efficient cause of pneumothorax in 90 *per cent.* of the cases in which air is found in the pleura (WALSHE, FULLER). Cases, also, are on record of pneumothorax ending in recovery, with remarkable absence of bad symptoms. Such a case has been described by Dr. Thorburn (*Brit. Med. Journal,* June 2, 1860), and is probably a unique example of pneumothorax occurring in a previously healthy man, and terminating in a recovery virtually complete, running its course without fever, and with singularly little pain or dyspnœa. Dr. Gairdner, also, in commenting on Dr. Thorburn's case, observes that the acute and terrible symptoms so characteristic of pneumothorax in typical cases are occasionally absent individually, or at least not of diagnostic value absolutely. The disease, therefore, is one which may be overlooked in such cases where the symptoms

are so latent that the date of invasion cannot be determined; and instances of the occurrence of pneumothorax being marked by the severe sufferings caused by another disease are so common as to be now well known (GAIRDNER). Cases of phthisis which do not seem to depart from their ordinary course, yet during the course of which aggravations are apparent, are just the kind of cases in which we may find, on examination, the pleura of one side more or less filled with air, but in which no date can be assigned to the pneumothorax, which has thus been detected at a period more or less remote from its actual occurrence (GAIRDNER, l. c., p. 391). To detect the existence of any minute opening in the pleura after death, the side of the chest affected should be filled with water, and if the lung is then gently inflated, bubbles of air will escape if a perforation exists, however minute. It commonly occurs in the area comprised between the third and sixth ribs (WALSHE, FULLER, CHAMBERS), "motion being greater there than in any other part of the chest," and therefore the pleura is less likely to be protected by adhesions there.

Symptoms.—The signs of pneumothorax are marked deficiency of respiratory murmur, without dull percussion, and with metallic phenomena, such as "a clear, mixing click, as of water dropping into a well" (*tintement métallique*), audible with almost every inspiration, and succussion sound in a more or less well-marked degree. Nevertheless, "it is extremely difficult," as Dr. Gairdner observes, "to say precisely what, in the present state of science, constitutes complete evidence, in a clinical sense, of the existence of air in the cavity of the pleura. The combination of *very marked* deficiency of respiratory murmur, with very marked euphonic respiration or metallic tinkling, would probably be accepted by the sceptical critic."

These phenomena only present themselves when the pneumothorax is very extensive; and such cases rarely occur except in conjunction with pulmonary tuberculosis, when it is generally fatal. "Pain may be only moderate in degree, or not characteristic. Dyspnœa may be merged in a more chronic affection of the chest; fever may hardly be observed amid the hectic of phthisis or of empyema; while the more special sensation of rupture, alluded to by some authorities, and the suddenly-felt rush of fluid or of cold air into the chest, as described by others, are certainly quite as often absent as not" (GAIRDNER, l. c., p. 387).

The signs of water, as well as air in the chest, may be discovered within a few hours after the presence of air has excited sufficient irritation to establish the pleuritis and serous effusion.

"The general symptoms in typical cases are comprised in physical distress and mental anxiety, as expressed in the countenance. The complexion is pale and dusky, and the lips more or less livid. The voice is weak; the skin moist, and often covered with a cold, clammy perspiration; the pulse is quick and feeble; and the respiration is extremely hurried" (FULLER, l. c., p. 204).

The physical signs in typical cases comprehend—

"Convexity of the affected side, with obliteration, widening, and even bulging of the intercostal spaces, immobility or diminished movement of the chest-walls, and inaction or diminished movement of the intercostal muscles, contrasting forcibly with the increased play of the opposite side of the chest and the energy of its intercostal action. *Palpation* informs us that *vocal fremitus* is diminished or altogether annihilated; that the intercostal spaces are more than usually elastic or resilient; and that the heart is more or less displaced. *Mensuration* confirms our impression respecting enlargement of the affected side, and the increased width of the intercostal side. *Percussion* elicits a clear tympanitic resonance, which sometimes changes its character, and becomes amphoric, and of a metallic quality over the trachea and larger bronchi . . . and the area of clear resonance on percussion may extend considerably beyond its normal limits, as the mediastinum, the heart, and the diaphragm are more or less displaced. . . . *Auscultation* furnishes different results, according as the amount of the effused air is larger or smaller. If the quantity of air be small, the respiration-sounds are weak and distant, and the vocal resonance is weak; if it be great, so that the lung is thoroughly compressed, the respiratory sounds and the vocal resonance are almost or altogether absent, except in the interscapular region, at the root of the larger bronchi, where diffused-blowing respiration, and diffused but loud vocal resonance, may still be audible" (FULLER, l. c., p. 204).

Certain signs resulting from the co-existence of air and serous or other fluid in the same cavity, which are not met with either in pleurisy or pneumothorax when they exist independently of each other, occur with the combined condition named *hydro-pneumothorax*, and are characteristic of it. These are described by Dr. Fuller as follows:—(1.) *Fluctuation*, which is felt by the

patient as well as by the observer, when the patient's body is abruptly jerked or shaken; (2.) A ringing, splashing sound—the succussion sound of Hippocrates—which is heard under the same circumstances; (3.) A remarkable metallic tinkling, which sometimes accompanies succussion of the patient, but which is also apt to accompany cough or inspiration, or a sudden change in the patient's posture (l. c., p. 205).

Prognosis.—When it affects the whole, or nearly the whole, of one side in tubercular disease, it is usually fatal after periods varying from minutes to weeks. *Limited* pneumothorax, however, is less fatal, as in cases, for example, in which the lung gives way by a mere *pin-hole perforation*, but the pre-existence or rapid formation of adhesions limits the escape of air to a part only of the cavity, and the pin-hole opening is sealed up. Pleurisy in such cases, therefore, in relation to pneumothorax, is not to be regarded as a fatal complication, but as a healing power (GAIRDNER, l. c., p. 396), the formation of adhesions often anticipating perforation of the pulmonary pleura in cases of tubercular phthisis. These adhesions limit the escape of air, prevent the utter collapse of the lung when air does escape, and so maintains to some extent the function of the lung, which otherwise would be destroyed as a consequence of such collapse. Prognosis is most unfavourable in perforation cases resulting from disease in the tissue of the lung, as contrasted with the more favourable prognosis in traumatic cases, in which the chest-walls are wounded, but in which the lung is not wounded. And those cases, *cæteris paribus*, are most apt to run an untoward course where there is great accumulation of air and great displacement of the thoracic organs (FULLER).

Treatment.—Puncturing the chest-walls, to relieve tension, may give temporary relief, by allowing the air to escape; but as the relief is only temporary, the operation ought only to be done in cases where the dyspnœa is urgent, and the displacement of the viscera such as demands that relief which the operation is calculated to give. Otherwise, the treatment of pneumothorax is best conducted by small doses of morphia—*i. e.*, stimulant doses—frequently repeated, combined or not by *æther*, or *alcohol* in small doses, with a view to overcome collapse, relieve dyspnœa, and subdue pain. When re-action follows the collapse, as indicated by heat of skin, strength and hardness of the pulse, soreness and pain of the affected side, local blood-letting by leeches, with saline

aperients, may be used if the strength of the patient warrants such a line of treatment. Turpentine and poppy fomentations, followed by blisters, are also indicated if life is sufficiently prolonged after the primary inflammatory symptoms are subdued. In other respects the treatment is similar to that stated under pleurisy.

Diseases of the Trachea and Bronchi.

BRONCHITIS.

Latin Eq., *Bronchitis;* French Eq., *Bronchite;* German Eq., *Bronchitis;* Italian Eq., *Bronchitide.*

Definition.—*Inflammation of the air-passages leading to the pulmonary vesicles, characterized by hoarseness, moderate cough, heat, and soreness of the chest anteriorly—symptoms which are more and more intense according to the severity of the disease. The natural mucous secretion is at first arrested, but subsequently it becomes increased in amount and altered in quality, tending to assume the corpuscular character.*

Pathology and Morbid Anatomy.—The mucous membrane lining the bronchial tubes may undergo the inflammatory process, followed by results peculiar to the texture of the part affected.

In diffuse bronchitis we find the inflamed portions of the mucous membrane of a deep venous red, and this redness may be general or partial, in spots, streaks, or arborescent forms. The more asthenic the inflammation, or the more feeble and cachectic the patient, the more livid and purple is the redness. The secretion of the tubes, at first arrested, is eventually increased in quantity and variously altered in quality. It becomes thin, watery, and frothy, and subsequently thicker and more consistent, assuming the appearance of pus. (See vol. i., pp. 80-90.) Abortive or young epithelial cells, loaded with serous effusions, and losing their vital cohesion with the basement membrane, are rapidly and easily discharged, constituting the thin, watery, frothy, serous expectoration of bronchitis in its early stage. Fibrinous exudation subsequently abounds, and the expectoration becomes tenacious and more opaque, and even pus-like.

Casts of the bronchial tubes.—In a very few cases lymph is thrown out, forming a false membrane or cast of the bronchial tubes. (See under "Characters of Sputa.") Since Dr. Baillie

first described and figured these tubular expectorated products, cases have been minutely described by many observers, and especially by Dr. T. Peacock, of St. Thomas's Hospital, in the *Transactions of the Pathological Society of London.* Figures of such casts are also to be seen in the description of a case published by him in the *Medical Times and Gazette* for 1854, p. 659. Such a form of bronchitis is known and described by the various names of *plastic bronchitis, bronchitis crouposa,* or *bronchité pseudo-membraneuse.*

Occasionally it has occurred that the expectoration of bronchitis is of a very fetid character, so that the case simulates gangrene of the lungs. An instance of this description (with other analogous cases) has been carefully described by Dr. Laycock, Professor of the Practice of Medicine in the University of Edinburgh. A chemical analysis of the expectoration in this case demonstrated the presence of *butyric* and *acetic acids;* and the odour was characteristic of the *butyrates of ethyl.* It resembled the smell of the May-flower or apple blossoms, but was combined with an odour of fæces (*Med. Times and Gazette,* May, 1857, p. 480). This condition is generally associated with dilatation of the tubes.

Ulcers.—In a small number of cases ulceration of the bronchial membrane occurs from within outwards, frequently associated with dilatation of the bronchial tubes, and constituting characteristic bronchial abscesses.

Many authors affirm that the bronchial membrane, when inflamed, is thickened, and more particularly at the points of division of the tubes, and that the various abnormal sounds depend on the degree of thickening of this membrane, slight alterations of diameter producing great alterations of sound. The thickening of the mucous membrane of the smaller bronchi may be so great as to cause a complete obstruction (ANDRAL). This thickened state of parts, however, is very difficult to demonstrate, and the different sounds so often heard in bronchitis are with much more reason ascribed to spasmodic contraction of the circular fibres; and there are good grounds for believing that a partial spasm of the tubes is in all cases connected with bronchitis, especially in its early stages; and which is also the chief cause of the narrowing of the tubes at particular points, in connection with sonorous and sibilant râles.

The bronchitis may affect one lung, or both lungs, or a part of a lung, and the upper lobes are more commonly affected than the lower ones. The larger bronchi are also supposed to be more commonly inflamed than the smaller ones. Hence it is that the more marked primary effects of bronchitis are most obvious in the bronchi towards the roots of the lungs; while the secondary effects which are apt to follow a prolonged or severe attack, such as *vesicular emphysema*, are mostly developed towards the pleural surfaces of the lungs, and especially towards those surfaces most directly under the influence of the expansion of the more moveable portions of the thoracic parietes.

A result of bronchitis which in its morbid anatomy may be mistaken for the tubercle of pulmonary phthisis has been recently demonstrated by Zenker, of Dresden. It is described as catarrhal induration of the pulmonary air-vesicles, by the accumulation of epithelium carried into them from the adjoining smaller bronchi, and which eventually fills up and consolidates the vesicular structure of the lung where this takes place. Zenker is one who does not believe in the possession of epithelium by the air-vesicles; and the tortuous vessels seen in them like varicose veins, especially in *stenosis* of the mitral valve, he regards as the normal state of vessels which are naked and uncovered by epithelium.

One most direct, invariable, and important result of bronchitis in the adult is condensation of the vesicular substance of the lung, of a peculiar type, as a result of mucous or other obstruction in the air-tubes leading to the condensed portion. Professor W. T. Gairdner has shown this, and that such a condensation is most apt to occur in bronchitis associated with asthenia, although in such cases the amount of obstruction may be small, as in patients debilitated by disease or by age. Such condensation is also produced by collapse of the pulmonary vesicles, the boundaries of this local condensation being distinctly mapped out by the interlobular divisions of the pulmonary lobules implicated. Such collapse, when extensive and sudden, is not only a frequent cause of death, but at the same time it is a fleeting, temporary condition of immense frequency, and important practically to distinguish from the condensation of pneumonia.

The morbid anatomy of bronchitis, which an examination of the air-passages discloses, shows that obstruction in the tubes

may be due to fluid mucus, or even to solid fibrinous coagula, or more or less prolonged spasm of the circular muscular fibre, and that such obstructions tend to the production of pulmonary collapse; and if the obstruction be considerable, and persistent, large portions of the lung may be emptied completely of air in the course of a few hours. The mechanism by which a mechanical obstruction, such as mucous fluid or more solid substances, brings about this collapse, appears sufficiently obvious when it is observed that such obstruction is never absolutely complete. The air gradually finds its way out by the edges of the obstructing substance, impacted as it is more or less in a series of diminishing tubes such as the bronchi, and acting the part of the ball-valve of a syringe, so that when the obstruction is driven onwards towards the narrower tubes by the force of inspiration, occlusion is more or less perfect only in the one direction. The expiratory force, however, so long as there is air in the vesicles, constantly tends to dislodge the obstructing body by pushing it towards the wider end of the tube. While, therefore, the entrance of the air is constantly and more or less effectually opposed, its exit is always permitted, so that ultimately the air-vesicles of the tubes beyond the seat of obstruction become completely emptied, and they collapse. The result of such collapse is a condensation of the tissue of the lung—a condition which had previously been ascribed to a limited inflammation of the pulmonary tissue, known as *lobular pneumonia*, and which was commonly believed to be peculiar to infants. Such collapse and condensation of the lung, however, whether in the lobular or diffused form, is an exceedingly common lesion in the adult, as shown by Dr. West and confirmed by Dr. W. T. Gairdner, especially in old persons, in typhus fever and in fatal dysentery, and is always associated with a certain degree of bodily weakness. Dr. Gairdner has further shown, however, that it is almost invariably found as a concomitant of fatal bronchitis, and that it depends on the obstruction of the tubes, as just described. When this collapse becomes permanent, it leads to still more obvious and important results:—

1. Like other parts which become useless, the collapsed portions become atrophied, leaving only a small amount of fibrous tissue in its place, the proper and special elements of the pulmonary tissue having disappeared. Such an atrophy causes a diminution

of the volume of the lung at the place where the collapse occurs.

2. By a definite law (which may be expressed thus:—*That a compensation by increased volume in one or more parts of the thoracic viscera invariably occurs to make up for diminished bulk in another, the internal capacity of the chest remaining the same*) this pulmonary collapse and atrophy invariably leads to vesicular emphysema of the lung, and may even tend to dilatation of the heart itself. The most constant result of collapse, however, is emphysema, so much so that the one almost never occurs without the other.

Bearing upon these statements, several general phenomena have been noticed, which may be thus shortly enunciated:—

1. That emphysematous portions of lungs are almost invariably free from every diseased appearance except the dilatation of the air-vesicles, and the consequent stretching and disorganization of their parietes.

2. That the bronchi leading to them are usually quite free from obstruction.

3. That vesicular emphysema by *increase of volume* of those portions of the lung to which the air has access, compensates for the *diminished volume* of the collapsed portion.

4. That vesicular emphysema prevails in the opposite parts of the lung to those in which the direct effects of bronchitis are observed.

The following are the forms of bronchitis which may be clinically recognized:—(1.) *Acute bronchitis* (a.) *of the larger and medium-sized tubes*—(b.) *Capillary bronchitis, and of the tubes generally—the "peripneumonia notha" of the older writers;* (2.) *Chronic bronchitis;* (3.) *Plastic bronchitis;* (4.) *Mechanical bronchitis,* such as knife-grinders' disease—*carbonaceous bronchitis,* or black phthisis; (5.) *Bronchitis secondary to general diseases,* such as typhoid fever; (6.) *Bronchitis secondary to blood diseases;* (7.) *Syphilitic bronchitis.*

General Symptoms of Bronchitis.—Bronchitis, of whatever kind, is generally preceded by fever, but more commonly by symptoms of what is commonly called "a *cold,*" or "a cold in the *chest.*" It often commences without any previous illness, and the uneasy sensations, frequently commencing about the region of the frontal sinuses, gradually pass down the nasal mucous passages, and

thence by the trachea and windpipe, are experienced in the chest, especially over the anterior region. The symptoms of bronchitis becoming developed are expressed by the hoarse, altered voice, the cough and expectoration, and are too palpable to allow us to mistake the nature and existence of the disease. In a very few instances of diffuse inflammation, especially in *capillary bronchitis*, the cough is dry and without expectoration; but far more generally it is accompanied by sputa. The sputa vary greatly according to the different degrees of inflammation, or according as that inflammation is acute or chronic, sthenic or asthenic. In acute cases it is at first a thin mucus, sometimes streaked with blood, then more opaque, and lastly purulent; in more chronic cases it may be merely a muciform saliva, or a gelatiniform mass; or it may be like the unboiled white of egg, so tenacious that it may be poured from one vessel into another without separating. In other instances it is puriform, varying from a laudable pus to a red or green putrilage. When purulent, it is usually formed into sputa, but in a few cases it is thrown up in large quantities unmixed, as from an abscess. The quantity of matter expectorated also varies greatly; sometimes only a few sputa in the morning, at others half a pint or a pint in the twenty-four hours, while other patients actually die suffocated from the immense quantity which is suddenly poured out, causing obstruction of tubes and collapse of the vesicular structure of the lungs.

The cough is seldom accompanied by any pain in the inflamed membrane, and has many degrees of violence. It may occur in paroxysms, and the sputa be discharged after a violent effort, at night or in the morning, or at other definite intervals. Again, it may be incessant, harassing the patient at every instant, causing a sense of soreness or constriction of the chest, and sometimes severe pain at the ensiform cartilage, in consequence of the mechanical exertion of coughing.

The urine of bronchitis varies greatly, as the grades of the disease are almost infinite, from a very slight affection of the larger tubes to a disease involving all the smaller tubes of both lungs, accompanied, perhaps, by collapse of the air-cells in some cases, and impeding aëration to a great degree in all. In these cases the urea is in large amount, and the pigment is increased, while the chloride of sodium is sometimes as deficient as in the

height of extensive hepatization; and it appears to Dr. Parkes that those cases in which the urinary ingredients are in extremely small amount are more commonly those of severe and general capillary bronchitis. The retention or non-excretion of the urinary substances seems to be more common in diseases attended with a considerable impediment to aëration than in other affections in which the absorption of oxygen is presumably less interfered with (*On the Urine*, p. 282).

With respect to the effects of the cough on the constitution, the patient, supposing the disease to be unconnected with any morbid poison or organic affection of the substance of the lung, suffers little in his general health, and often feels he would be well if he could get rid of "the cough." In other cases he loses flesh, ejecting every meal, from the violence of the cough, or he sinks into a state of marasmus simulating phthisis. His pulse is generally natural, although in some cases it is frequent; his bowels also are regular. In bad cases, however, the patient's nights are broken, and he sleeps towards morning; while in slighter cases he sleeps through the night, but is disturbed early in the morning by cough and expectoration.

The duration of bronchitis is uncertain. It sometimes terminates in a few hours, sometimes in a few days, ceasing with the cold weather that ushered it in. In other cases its duration is long, and it is with difficulty recovered from; thus often laying the foundation of other formidable diseases which may ultimately destroy the patient. In old persons it generally returns every winter, or lasts, with intermissions, during the whole year.

Physical Symptoms.—The natural and healthy respiratory *bruit* of an adult has been compared to the sound heard during the calm sleep of a healthy child. In bronchitis this sound in the adult is changed, and varies, in different cases, from a tolerably sharp sound, which, when multiplied from a number of bronchi similarly diseased, resembles a chirping sound, or the bass notes of the violoncello. The sounds thus embrace a musical scale of considerable compass, the principal and more marked division of which compose the *sonorous* and *sibilant* râles, as they are termed. The cause of the higher notes has been supposed to be owing to a thickening of the mucous membrane at the orifices of the various bronchial tubes, so that the natural *embouchure* is

narrowed, and a musical wind instrument is thus formed. To those who have observed in the dead body a swollen state of the bronchial membrane, this explanation may seem satisfactory; but to those who have not seen such a phenomenon, it seems more easy to explain this morbid sound by the different degrees of contraction of the circular and longitudinal fibres of the bronchi, in the same manner as we observe contraction of the muscular fibres of the œsophagus, or of the small intestines, causing a constriction. Besides the alteration of tone of sound in bronchitis, its quality is also often affected by the presence of liquid matters within the cavity of the bronchi; and hence we have it interrupted and modified by the air passing through bubbles of mucus; and as the size of these bubbles, and their viscidity, vary, so the sounds vary. Hence a scale was established by Laennec, whose extremes are the "râle muqueux," and the "râle tracheal;" the former representing the bursting of small slightly viscid bubbles, the latter larger ones of greater tenacity, and yielding a gurgling sound. Sometimes this mucus, instead of being fluid, hardens so as occasionally to adhere and play as a valve, giving rise to a clicking noise. These are the various morbid sounds heard in bronchitis; and the danger of this disease is denoted by the quantity of fluid effused, and may be judged of by the nature of the sound. The sharp chirping sound is more to be feared than the graver and deeper notes; for grave sonorous notes originate in the larger tubes, chirping, whistling notes in the smaller; and the danger in bronchitis increases in proportion as the finer bronchial tubes become involved (*Edin. Med. Journal*, 1864, p. 1114). When the sounds of expiration are also much prolonged, severe bronchial inflammation is indicated.

Percussion generally returns a healthy sound in bronchitis. An important physical sign is one which indicates a sudden disappearance of the respiratory murmur over a definite part or parts of the lung. Percussion sometimes shows, however, that the part still contains air; and therefore it is presumed that the disappearance of the murmur is due to obstruction of one or more of the bronchial tubes; and which, as already shown, may lead to *collapse*, *condensation*, *atrophy*, and *emphysema*. In some cases the respiration is greatly accelerated, varying from 36 to 50 or 68 in a minute, especially in cases of capillary bronchitis. The pulse at the same time rapidly loses its strength, and becomes excessively

frequent—120 to 150. The ratio of the respiration to the pulse is therefore greatly altered from the normal standard—that is, about 4 or 4½ beats of the pulse for every respiration; and thus *pulse-respiration* ratios of 3·0, 2·5, or 2·25 to 1 are not uncommon.

Diagnosis.—It is hardly possible to confound bronchitis with any other disease; but there is often much difficulty in assigning its cause and distinguishing it from phthisis. The absence of great emaciation, and the clear resonance returned on striking the chest, are the most salient points in diagnosis.

Treatment.—When the symptoms of a "common cold" first express themselves, and even when the sensations have extended to the chest, as indicated by the hoarseness and tendency to cough, the disease may be at once subdued in a healthy person by a full stimulant but not narcotic dose of opium or morphia (i. e., *one grain of opium, or a fourth of a grain of morphia*) at bedtime; or by an alcoholic diaphoretic drink; or by *five* grains of *carbonate of ammonia*, or *ten* to *twenty* grains of *muriate of ammonia;* or, if the appetite is unimpaired, a full supper, followed by a moderate amount of some hot alcoholic stimulant, may have the same effect.

When the attack is sudden, and attended with marked depression, associated with aching pains in the head, back, and limbs, and much febrile disturbance, opium, if taken early enough, is a perfect remedy. "One-third of a grain of muriate of morphia ought to be taken at bedtime, and its influence will begin to be felt in from twenty minutes to half an hour, by the gradual disappearance of the sense of intense weakness, the relief of the pains, and that peculiar feeling of thorough and evenly distributed warmth of the whole body which is so different from fever on the one hand or chilliness on the other" (ANSTIE, *op. cit.*, p. 120). Natural sleep ought to supervene, and the morning ought to find the patient well.

If such remedies are delayed too long, the object to be aimed at next is to induce a copious perspiration and a continued action of the skin and kidneys, in the first instance, by small doses frequently repeated, of *antimonial* and *ipecacuanha wines*, or *nitrate of potash*, or *acetate of potash*, as well as *bicarbonate of potash* and *aqua potassæ* combined at a much later period with tinctures of *squills* and *hyoscyamus*.

In the early stage of the disease we have seen that the

bronchial membrane has its normal moist condition altered to a very dry state; and the object of treatment is to bring about a return of the naturally moist condition. This is best effected by the inhalation of the vapour of hot water, the use of *tartar emetic* in doses of *one-twelfth* to *one-sixth* of *a grain*, the use of *ipecacuanha* in *one-quarter or half grain doses;* and these may be aided by *hemlock, henbane, aconite,* or *hydrocyanic acid* in appropriate doses. From the frequent inhalation of steam great benefit is derived (EASTON).

Abundant experience has shown that general bleeding in acute *bronchitis* uniformly weakens the patient, without greatly influencing the disease. Neither has medicine any very marked effects in the cure; for although some persons rapidly get well under a given treatment, yet many similar cases, under exactly the same treatment, will run on for weeks, and perhaps for months, without any amendment. In the most acute cases of bronchitis, however, some blood may be taken from the chest, with great caution not to take too much, either by cupping between the shoulders or by leeches, and in general from ten to twelve ounces are sufficient. It is only in cases of congestion of the brain, heart, or venous circulation threatening asphyxia, that general blood-letting is imperative. Next to blood-letting, *tartarized antimony* administered in solution in doses of *a sixth* or *a quarter of a grain, every three or four hours,* conduces to free secretion, and generally to mitigate the symptoms of the disease. Dr. Fuller regards *digitalis* as a useful adjunct to the antimonial treatment; and the air-passages should be fomented by the inhalation of moist warm air, as by the steam of hot water, the secretions being at the same time stimulated, so that the bowels act freely. When the expectoration becomes thicker and less copious, the antimony may be decreased, and *squills,* or *ipecacuanha,* with *paregoric,* given. After this a blister should be applied to the chest; and on its being removed, a large lintseed poultice should be placed over the blistered part, and be continued for many hours, which will not only keep the ulcerated surface open, but gratefully foment the part and relieve the patient. The bowels should be freely evacuated by a purgative dose of calomel, combined with *compound jalap powder,* and they should subsequently be kept in regular and gentle action by some neutral salt, such as the *sulphate of magnesia* in the *liquor*

ammoniæ acetatis, combined at the same time *with some nitrate of potass.* The *compound jalap powder* is a most useful remedy when the system will bear it. Œdema is greatly relieved by its use, whether of the lungs or of the body generally. The neutral *citrates, tartrates*, or *acetates* of the alkalies are useful eliminating remedies.

After these means have subdued the severity of the symptoms at the outset, expectoration should be promoted by such remedies as *squills, ipecacuanha*, and *tartar emetic*, combined with *hyoscyamus* or *conium*. Opium in narcotic doses (*i. e.*, above a grain) is inadmissible if the evacuation of the loaded air-passages is to be promoted, because its tendency, in a narcotic dose, is not only to diminish the secretion, but to paralyze the action of the mucous passages in eliminating that secretion. When large doses of opium have been given at this stage, death has been known to follow, and necroscopic examination has revealed the air-passages loaded with frothy serous mucus, and the air-cells congested and collapsed. It is not till after secretion has begun to diminish, in acute cases, that opium may be prescribed with benefit in stimulant doses, and it is then to be given in the form of the solution of the salts of morphia, added at bedtime to the doses of the *cough mixtures* so usually administered. If the disease shows a disposition to pass into the chronic stage, *quinine*, with *squills* and *conium*, may be administered, or a draught containing full doses of *cinchona, with five grains of carbonate of ammonia*, or *ten to twenty grains of the muriate of ammonia, and thirty or forty minims of the compound tincture of benzoin or of the balsam of Peru*, will generally facilitate expectoration and relieve the dyspnœa. If a "*hearty cough*" is not attended by easy and free expectoration, but the chest remains loaded, powerfully stimulant expectorants may be given in aid of other remedies, such, for example, as the *decoction of senega* or the *mistura ammoniaci* (Fuller). The patient throughout the treatment should remain in a room where the air is kept moist by the evaporation of boiling water from large flat dishes near the bed, and the temperature of the air should be maintained at 63° to 68° Fahr.

In chronic cases of bronchitis, especially in patients "who have made considerable progress in the journey of life, a lower tone of the system generally prevails, and a greater laxity of aërian mem-

brane—particularly with excessive secretion, often muco-purulent" —characterizes such cases, and in them, after blistering, and perhaps poulticing the chest repeatedly with mustard poultices, the treatment in general should be more tonic. The *camphorated mixture* or *paregoric* and stimulant expectorant remedies are indicated for occasional but not for constant use. It was in these cases that the late Dr. Easton, Professor of Materia Medica in the University of Glasgow, so well showed the necessity of using remedies which will promote expectoration of the secretion which accumulates in the bronchial tubes. Such remedies are those which tend to invigorate the general system. Besides the selection of a beneficial climate, and the use of nourishing, easily-digested food, stimulating embrocations may be rubbed not only over the chest, both before and behind, but along the sides of the neck.

In the Meath Hospital at Dublin a liniment composed of the following ingredients is extensively employed by Drs. Graves and Stokes, and is recommended by them, as well as by Dr. Maclachlan:—

℞. Spt. Terebinthinæ, ʒiii.; Acid. Acet., ʒiv.; Vitelli Ovi, i.; Aq. Rosæ, ʒiiss.; Ol. Limon., ʒi.; *misce.*

As a rubefacient, it is to be applied morning and evening, when it generally reddens the skin and produces small pimples. In several cases the secretion of the kidneys is increased during its use (MACLACHLAN).

Of tonic remedies, which are invaluable, my friend the late Professor Easton, from his extensive experience, put most reliance on the influence of *nux vomica, iron, and cinchona.* He was in the habit of prescribing them in the form of a syrup composed of the *phosphates of strychnia,* of *iron,* and of *quinia,* so that, in doses of a tea-spoonful three times a day, each dose shall contain the *thirty-second part of a grain of phosphate of strychnia* and *one grain respectively of the phosphates of iron and quinia.* (Formula for the preparation of this compound, see page 62, *ante.*)

Combined with these remedies, the inhalation of *slightly* irritant vapours has a beneficial effect, as of *vinegar, turpentine, chlorine, and iodine.* Inhalation of vapour is often unsatisfactory, on account of the difficulty of getting an apparatus to hold a sufficiently large volume of boiling water. In the practice of my

friend Dr. Fergus, of Glasgow, I have seen an admirable arrangement for inhalation, which overcomes this difficulty. It consists of a globular glass flask, about eight inches diameter, and six inches deep, having a wide mouth, a closely-fitting cork, carrying an inlet tube which descends to the bottom of the vessel, and an outlet tube, to which a flexible mouth-piece is fixed. It is used as an Eastern uses a *nargilé* for smoking through water.*

Of the fetid gums, *ammoniac* in particular is a useful remedy. An emulsion of *gum* ammoniac in *diluted nitric* acid is a combination from which decided beneficial results are obtained. Professor Easton gave the following formula for its prescription—namely, *an hundred and twenty grains of the gum ammoniac dissolved in two fluid drachms of diluted nitric acid and twelve ounces of water*, compose a mixture of which an ounce may be given in gruel three times a day (*Glasgow Med. Journal*, Oct. 1, 1863). On the contrary, it is often advantageous to administer astringent remedies, and one of the most useful is *tannic acid* in doses of one to three grains two or three times a day, as originally recommended by Dr. Alison, of Edinburgh; or the *oil of cubebs* to the extent of ten drops three or four times a day on a piece of sugar.

Acute bronchitis is very apt to be latent in old people, and to be complicated with gastric or gastro-enteric inflammation (MACLACHLAN). The treatment must therefore be modified to meet such a contingency. When there is tenderness at the pit of the stomach, nausea, and failure of the appetite, with the general condition approaching a typhoid state, in persons beyond the meridian of life, the pectoral symptoms are often apt to be marked by such associated disorder. The stimulants of food (by *enema* in the form of soup, if unable to be taken by the mouth) and of alcohol are the main remedies necessary from the very beginning. Abstinence cannot be enforced with safety. Leeches must be applied to the pained gastric region.

When the disease is associated with a tendency to gout, colchicum must be given. "It allays the cough, promotes the flow of urine, keeps up a regular alvine discharge, and can be given much more generally than squills, because it does not produce

*These inhalers are made of glass, so prepared as to receive boiling water without breaking; and are fitted up with tubes ready for use by Mr. Hugh Reid, chemist, 423 Argyle Street, Glasgow.

that feverishness which results from the use of the latter remedy, and can therefore be employed where there is considerable fever" (FORBES). It requires to be administered with great caution in the aged and infirm (MACLACHLAN).

In the protracted bronchitic affections of the aged, diuretics are of great service; and the following formulæ are recommended by Drs. Maclachlan and Stokes, as well suited in a variety of cases of senile chronic catarrh:—

℞. Decocti Senegæ, *f*℥vij.; Potassæ Nitratis, gr. iii.; Tinct. Camph. Comp. vel Tinct. Conii, ♏xx.; Spiriti Ætheris Nitr., *f*℥ss.; Oxymellis Scillæ, *f*℥ss. Fiat haustus ter die sumendus (MACLACHLAN).

℞. Liq. Ammon. Acet., ℥iii.; Potassæ Acetatis, gr. xx.; Aceti Scillæ, *f*℥ss.; Spirit. Æth. Nitr., *f*℥ss.; Tinct. Camph. Co., ♏xx.; Mist. Camph., ℥vi.; Syrupi Aurant., ℥i. (MACLACHLAN). Fiat haustus ter die sumendus.

℞. Decocti Senegæ, ℥v.; Tinct. Camph. Comp. Scillæ, ā ā ℥ii.; Syrupi Tolut., ℥iv. Sumat ℥ss. vel. ℥i. ter die (STOKES, MACLACHLAN).

When gastric irritation prevails, the administration of balsams, gum resins, and terebinthine remedies must be suspended.

DILATATION OF THE BRONCHI—SYN., BRONCHIECTASIS.

LATIN EQ., *Dilatatio;* FRENCH EQ., *Dilatation;* GERMAN EQ., *Erweiterung;* ITALIAN EQ., *Dilatazione.*

Definition.—*A cylindrical, or fusiform, or saccular dilatation of a bronchial tube, or of several tubes, at one or more points, or through a considerable portion of the tubes.*

Pathology.—The true bronchiectasis arises quite independently of all other pulmonary affections; but there are several minor varieties which result from other changes in the lungs, such as from *hooping cough; suffocative capillary bronchitis; stricture of bronchi; long-standing indurations of lung substance, tubercular or inflammatory; the remains of chronic tubercular cavities,* or *abscesses in the lung tissue.*

The most important forms of the *true bronchiectasis* are—(1.) *The general or uniform,* in which there is a cylindrical or fusiform dilatation of a tube, or of several tubes throughout considerable stretches of their extent; (2.) *The saccular,* or ampullary, in which there occurs an abrupt dilatation of a tube at a particular point, or at several points. When dilatations exist in

neighbouring bronchi, communications may become established between them, and to such an extent that several bronchial tubes open into one common cavity. Wasting of the muscular and elastic coats is a usual result, and there is degeneration of their tissue; and in consequence the dilated tubes open into each other without any truly ulcerative process, as shown by Dr. T. G. Stewart, from whose excellent description of "*Dilatation of the Bronchi*," in the *Edin. Med. Journal* for July, 1867, this account of *Bronchiectasis* is taken. Copious yellow or inspissated mucus, sometimes with casts of tubes, and often very fetid, exists in the dilated tubes, associated with crystalline fats and fungi. In some cases the tissue of the lung is condensed, from cirrhosis or fibroid degeneration of the lung, which may proceed to more intense induration, ulceration, and even gangrene. The site of *bronchiectasis* is usually the lower lobe and the middle lobe of the right lung. It occurs towards the apices sometimes. Usually it affects many bronchi and occurs in both lungs. The essential element of the lesion in bronchiectasis is atrophy of the bronchial walls, which rapidly yield to the pressure of air. The enfeebled and dilating condition of the bronchi favour the accumulation of the mucus secreted by the mucous membrane, which, accumulating and undergoing decomposition in the dilated cavities, leads to inflammation and the formation of villous processes, to increase of connective tissue, and to further consolidation of surrounding lung.

Symptoms and Clinical History.—The disease comes on insidiously; but gradually symptoms of bronchitis become well marked. Breath and sputum become fetid, and general health is impaired. Decomposition of the secretions is followed by lung consolidation, ulceration, abscess, or gangrene. Perforation of the pleura, empyema, or pneumothorax may prove fatal; or death may result from exhaustion due to the constant discharge of the sputum. A peculiar febrile disturbance resembling *septicæmia* may also terminate life. Unfortunately, the tendency is to a fatal result; but recovery may take place—1st. *From cretification of the contents of the dilated tubes, and the conversion of their walls into a sort of fibrous capsule;* 2d. *From penetration of the pleura and thoracic parietes and discharge of the contents outwards.* Dr. Stewart considers the disease as probably hereditary and constitutional.

The most characteristic symptoms are, *the odour of the breath*, the *characters of the sputum*, and the *cough*. The fetor of the breath is something distinct and different from that of gangrene, and is not present in all cases, and not till decomposition of the bronchial secretion has taken place. *Blood* is sometimes expectorated with the sputum, sometimes in large quantities, as in phthisis, at other times merely in streaks over the expectoration. *Cough* is frequent, and occurs in paroxysms, but is moist, soft, and usually quite painless; and after the fit of coughing, large quantities of the sputum are brought up with difficulty. Exertion induces dyspnœa. *Auscultation* gives varied sounds—cavernous, moist râles, sometimes even gurgling, and the site of these sounds aids to distinguish them from those of phthisis, also the history and progress of the case.

Treatment.—Opiates, to relieve cough; balsamic remedies (*tolu, tar, turpentine, copaiba, cubebs*), and astringents like *catechu* or *rhatany*, with the use of counter-irritants and the inhalation of variously medicated vapours, are all useful aids in ameliorating the condition of the patient. Inhalation of disinfectants capable of being so used, such as *creosote, carbolic acid, sulphur vapour*, and the like, are of special service.

Diseases of the Lung.

EMPHYSEMA.

Latin Eq., *Emphysema;* French Eq., *Emphyseme;* German Eq., *Emphysem;* Italian Eq., *Emfisema.*

Definition.—(1.) Vesicular Emphysema—*A misshapen enlargement of the air-cells of the lungs, by dilatation of them, attended with gradual effacement of the functional blood-vessels distributed over their walls; anæmia of the lung in the affected parts, tending to dilatation of the right side of the heart, with anasarca.* (2.) Interlobular Emphysema—*Air infiltrating the meshes of the connective tissue of the lungs.*

Pathology.—Some confusion has arisen from the use of the term "*Emphysema*" (which means the presence of air in the connective tissue) to designate dilatation of the air-cells of the lungs. Here the air is where it ought to be; but the air-cells are too large and misshapen, and contain too much air; and being limited in

this way to the vesicles of the lung, this form of disease was named by Laennec "*vesicular emphysema.*" In 1698 the disease was identified and well described by Sir John Floyer as existing in broken-winded horses; and Dr. Baillie, in subsequently describing enlargement of the air-cells of the lungs, refers to Sir John Floyer's description as applicable to the lungs of the human subject.

The dilated air-vesicles vary from the size of millet seeds to that of Barcelona nuts, or even larger; but when they form a great expansion, it is probable that many air-vesicles are dilated into one common cavity by rupture of the partitions which separate them from each other. The dilated vesicles may be seen clearly through the pulmonary pleura: they also protrude from the surface of the lung. The emphysematous parts are pale, and sometimes quite white; the tissue is drier than normal; it cannot be easily emptied of air, resembling the lungs of a reptile rather than those of a human being. It possesses fewer capillary vessels; and they become obliterated by distension of the air-cells. The lung is therefore anæmic, and contains less moisture than the normal lung. It is so dry and light that it floats much higher in the water than a healthy lung.

Causes.—Several theories have been put forward to explain the mechanism which produces emphysema. Dr. Elliotson considers a want of due expansion of the lungs as the most common cause of emphysema. "Whatever prevents any one part of the lungs from expanding when the thorax expands—whether it be a material obstruction of the bronchial ramifications, or a compression of them, or whatever else, it will occasion those parts which remain dilatable to keep dilated in a corresponding increased degree, in order to fill up the vacuum which the expansion of the chest occasions. When we inspire, we dilate the chest, and the air rushes down the trachea, and the lungs follow the dilated portions. If there be any part [of the lung] that will not dilate, then other parts are over-dilated to fill up the vacuum; and in that way those parts which we distend are *over*-distended, in order to compensate for the want of distension in other parts; and when once *over*-distended, they are often unable to recover themselves, just as is the case in other parts of the body—the urinary bladder, for example. I presume it is on this account that dilatation of the air-cells is so common in persons labouring under

chronic bronchitis, especially where the membrane is most thickened, and where the secretion, if there be any, is tough and adherent, so as to produce obstruction" (*Practice of Medicine*, p. 851). Dr. Watson follows Laennec in believing that the dilatation in the outset is mainly due to the imprisonment of air within the cells under the influence of disease, such as imperfectly obstructed tubes, so that air enters the vesicles more readily than it can escape from them. More and more air then accumulates, and is incarcerated in certain air-cells, which, yielding to the distending force, lose their elasticity and become permanently large.

But, to confirm this view, it ought to be shown that the dilated air-cells are those belonging to the tubes in which the obstruction exists; and Dr. Elliotson is of opinion that they are not those in which there is obstruction. Dr. Gairdner, indeed, has very ably demonstrated this in connection with the occurrence of emphysema in bronchitis (see page 705, *ante*). The disease forms one of the most serious complications of *bronchitis;* and the tendency to vesicular emphysema appears to be hereditary—60·4 *per cent.* acknowledging hereditary transmission (FULLER).

Symptoms.—In typical cases of emphysema the patient is short-winded, and distressed by a constant sense of fulness and oppression at the chest, and generally seeks advice after suffering and discomfort have become too great for him to bear any longer.

The difficulty of breathing is often aggravated by spasm, as in *asthma;* and emphysema is a frequent consequence of that disease —the one re-acting on the other, so that the phenomena of each are mutually aggravated.

The physical signs are, incompleteness of the act of expiration, the thorax remaining prominent and round over the emphysematous lung. In spare persons the clavicles are not well defined. Percussion, over the bulging parts especially, yields a peculiarly clear and resonant sound; and although there is thus shown to be abundance of air underneath the part which yields such a sound, yet the *vesicular murmur* of breathing is extremely indistinct, showing the air is not in motion there. It is shut up in the enlarged air-cells (WATSON).

The disease tends to impede the circulation through the lungs, and so to produce hypertrophy, with dilatation of the right side

of the heart, nervous congestion of the head and face, attacks of palpitation, paroxysms of cough and dyspnœa, œdema of the feet and legs, general anasarca; and dropsical effusion is a frequent termination of the disease.

Treatment.—Apart from the management of the bronchial congestion, on the principles already given under *bronchitis*, little can be done for the special treatment of *emphysema*. If bronchial spasm prevail, Hoffman's anodyne may give relief. It is the *spiritus ætheris* of the British Pharmacopœia, of which *thirty* to *sixty minims* may be prescribed in *camphor* water, or in *spiritus ammoniæ aromaticus*, or in *volatile tincture of Valerian;* or it may be combined with stimulant doses (*one grain*) of opium; or with *twenty minims* of the *æthereal tincture of lobelia, belladonna, conium;* or the *æthereal tincture of Indian hemp and hydrocyanic acid* may each in turn be found of service (FULLER).

Dry cupping between the shoulders often relieves passive pulmonary congestion; and if an attack is imminent, an emetic, or unloading the bowels by a dose of the *compound jalap powder*, may prevent its accession or moderate the paroxysm.

PNEUMONIA.

LATIN EQ., *Peripneumonia;* FRENCH EQ., *Pneumonie;* GERMAN EQ., *Lungenentzündung*—SYN., *Pneumonie;* ITALIAN EQ., *Pneumonitide.*

Definition.—*Inflammation of the true pulmonary tissue, which in its acute sthenic form, uncomplicated by constitutional or specific diseases, runs a definite course, expressed by severe febrile symptoms, which come on suddenly, attaining in a few hours a great intensity, and which undergo a no less sudden abatement or improvement between the fifth and tenth day, in proportion to the severity of the disease; and while the local productive results of inflammation in the form of the lung-lesion are yet intense, but which are eventually removed.*

The natural course of pneumonia is materially modified by constitutional or specific diseases, especially if any organ, such as the kidney, the heart, or the liver, is involved; or it may be modified by the secondary contamination of the blood by absorption of lung-exudation in the latter stages of the disease, tending to inflam-

mation of the other lung, to pleurisy, to pericarditis, or to blood-coagula in the cavities of the heart or great vessels.

Pathology.—In the continental cities of Europe about *eight per cent.* of all the deaths are caused by pneumonia; and while about *three per cent.* of all diseases are due to the same cause, about *two per cent.* of all cases of disease in hospital are referable to pneumonia. It is subject to more or less well-defined periodic fluctuations, and sometimes appears as if it were epidemic; while its prevalence has been observed to be very much coincident with that of *typhus* fever (Niemeyer, *New Syden. Society Year-Book*, 1862).

The phenomena which are appreciable during the natural course of a disease which is uncomplicated and unmodified by any other disease or by treatment, afford the best means of studying its pathology. To Dr. Parkes, the Emeritus Professor of Clinical Medicine in University College, science is indebted for the records of such a case (*Med. Times and Gazette*, Feb. 25, 1860). The case was one of acute sthenic pneumonia, which occurred in a well-built temperate man of about ten stones weight, and which Dr. Parkes conscientiously believed could be left without any treatment (beyond the application of a few leeches) to the unassisted processes of nature.

The characteristic phenomena of pneumonia in the majority of cases are evolved in a regular and consecutive manner; and they may be considered under the three following heads:—

1. *The Course of the Pyrexia as Measured especially by the Thermometer and the Pulse.*—In the case which Dr. Parkes records, the commencement of the pneumonia is dated from the *shivering;* and the patient was admitted to hospital on the *third day* of the disease, which was completed at four P.M. of Jan. 7, 1860. He was then intensely febrile, with flushed cheeks, constant cough, viscid bloody pneumonic expectoration, hurried breathing, with crepitation and bronchial respiration over the posterior base of the left lung. The conjunctivæ were a little yellow. Thermometrical observations in the axilla were taken hourly during the day—from nine in the morning till eleven and twelve at night—by Mr. Ringer and Mr. Miller; and the following Table gives the general results—records of the pulse and respiration being also given, for the purpose of comparison with the temperature and with each other:—

TABLE OF THE TEMPERATURE, PULSE, AND RESPIRATIONS.

FROM HOURLY OBSERVATIONS DURING THE DAY. MEAN NUMBER OF DAILY OBSERVATIONS,—14.

Date.	Day of Disease. (The disease commenced at 4 p.m.)	Temperature in Axilla. Hourly Observations during the Day.					Pulse. Hourly Observations during the Day.					Respirations hourly observed. Mean No.
		Mean.	Max.	Min.	Hours of Max.	Hours of Min.	Mean.	Max.	Min.	Hours of Max.	Hours of Min.	
Jan. 7.	Part of third and part of fourth day.	103·6° Fahr.	104·2°	102·6°	7 P.M.	9 A.M.	108	120	100	1 A.M.	2, 5, 10, and 11 P.M.	36
,, 8.	Part of fourth and fifth days.	103·9°	104·2°	103·6°	5, 7, and 8 P.M.	11 A.M.	107	116	98	7 P.M.	11 A.M.	36
,, 9.	Part of fifth and sixth days.	103·4°	104·2°	103·0°	1 P.M.	8 and 10 P.M.	106	112	100	4 and 6 P.M.	10 and 12 P.M.	43
,, 10.	Part of sixth and seventh days.	100·6°	101·4°	99·4°	9 P.M. 1 P.M.	11 P.M.	93	112	82	5 P.M.	10 P.M.	38
,, 11.	Part of seventh and eighth days.	98·6°	90°	98°	3, 5, and 6 P.M.	11 A.M.	76	84	72	2 P.M.	4 P.M.	30
,, 12.	Part of eighth and ninth days.	98·5°	98·8°	98·4°	1 P.M.	10 A.M., 2 and 4 P.M.	76	84	70	1 P.M.	9 A.M.	31

During the *third* and *fourth days* of the disease the temperature was uniformly high, the difference between the maximum

and minimum being only 1·6° Fahr. on the *third*, and 0·6° Fahr. on the *fourth day*. The *fourth day* was the most febrile, both as to mean temperature and as to the constancy of its height. The *fifth day* was scarcely less febrile, the mean temperature being only 0·2° Fahr. below the *fourth day*.

Towards the end of the *fifth day*, however, and during the commencement of the *sixth day* (from four to twelve P.M., 9th January), the thermometer decidedly fell slightly; then, during the early part of the *sixth day* of the disease (night of January 9), it made a great descent from 104·2° to 101° Fahr.; and after this time it never rose above 101·4°. During the whole of the after-part of the *sixth day* and the first part of the *seventh*, the temperature continued to fall, and at 11 A.M., January 11—the nineteenth hour of the *seventh* day of the disease—it reached 98° Fahr. In thirty-six hours it had fallen from 103·2° to 98°, or no less than five degrees. Afterwards it oscillated for two days between 98° and 98·8°, but never rose above this latter point. The fever, in fact, had ended by crisis (a sudden and rapid defervescence), which is usually accompanied by a strong action of some eliminating organ, such as the skin, the kidneys, or the bowels; and in the case recorded by Dr. Parkes there were both profuse sweating and considerable urinary excretion; but whether more at the period of rapid fall than before is uncertain.

The pulse ranged from 120 to 100 during the first three days, and then fell to 90, 80, and 70. On comparing carefully the hourly variations of the pulse and temperature, it is quite clear that there is a connection between them, so that either simultaneously or often a little before or after, a fall or rise in the thermometer occurred, with a fall or rise in the number of the pulse. Not unfrequently the alteration in the pulse occurred before the change in the thermometer; and sometimes the pulse rose, though never greatly, when the temperature was falling. When the thermometer oscillated and finally fell, the pulse fell at the same time, and very uniformly. The respirations averaged 38 in the minute during the first four days, and 35 afterwards. They did not fall nearly so much as the *temperature and the pulse;* and were not nearly so good an indication of the course of the pyrexia. As the mean of the thermometer was not above 104°, as the mean of the pulse was not 120, and as the mean of the respiration was not 40, the case must be considered a slight

one, according to the rule laid down by Wunderlich—namely, that the intensity of cases of pneumonia is to be judged of by the concurrence of these phenomena, so as to call all cases slight that fall *below*, and all cases severe that are *above* those averages in which *the temperature records* 104°, *the pulse more than* 120, *and the respirations more than* 40 *in the minute during the height of the disease.* It is only by a rigorous application of some such rule to the cases of pneumonia which are grouped together to furnish statistical data as a guide to treatment, that anything like trustworthy results can ever be obtained. Such results have not yet been so obtained; and it is scarcely to be expected that the task will be accomplished in our day.

2. *The Course of the Local Lung-Symptoms.*—When the patient was admitted, there was considerable crepitation, and some bronchial respiration in the lower lobe of the left lung. The hepatization increased, and was considerable on the fifth and sixth days, and its greatest amount was either at the period of the *defervescence* or was subsequent to it—the number of respirations being even greater after the temperature and pulse had commenced to fall than before, so that they appeared rather to run *parallel with the amount of the hepatization than with the general fever.* The sputa were most bloody during the *third* and *fourth days* of the disease—were less florid and more rusty on the *fifth* and *sixth*—after which they became less viscid and free from blood or hæmatine. The pain in the side disappeared on the *fourth* or *fifth day.* After the *seventh* or *eighth day* the bronchial respiration began to lessen, and had ceased by about the twelfth to the sixteenth day. Harsh respiration, some *redux* crepitation, and a little sonorous *râle* were left for some days more.

3. *The Condition of other Organs.*—Having twice had rheumatic fever (eleven and two years before the pneumonia), the heart of the patient in one or other of the attacks had been slightly damaged, and he had slight aortic obstruction, with mitral regurgitant disease, but without any marked alteration in the size of the heart's cavities. There was supplementary breathing in the right lung, and about the angle of the right scapula was some slight suspicious bronchial respiration, as if some consolidation were there; but if so, it disappeared early.

The liver was not enlarged nor tender, but the conjunctivæ were very slightly yellow; and the yellowness disappeared at

convalescence. There was thirst, loss of appetite, and dry, furred tongue.

The action of the eliminating organs was as follows:—(*a.*) The skin acted profusely throughout, both before and at the period of defervescence; (*b.*) The bowels were rather confined, but the motions were said to be natural; (*c.*) The amount of excretion from the lungs was undetermined; (*d.*) The excretion from the kidneys was not determined till during the *fifth* and *sixth days* of the disease, the last day of the intense pyrexia. On this day there was a little albumen (one-sixth of the height of the urine in a test tube). There were no chlorides, and on the following day the albumen had disappeared. The other urinary ingredients were not determined; but there were copious deposits of lithates on the *fifth, sixth, seventh, eighth,* and *ninth* days of the disease, and probably afterwards, of which no note was made. On part of the *seventh* and *eighth day eight grammes of chloride of sodium* were taken by the mouth, and on the *eighth, ninth,* and *tenth* days, as on previous days, there was no chlorine in the urine. But on the *eleventh* day of the disease the chlorine began to re-appear, was in some quantity on the *fourteenth* day, and on the *twenty-first* and *twenty-second* days of the disease it was so abundant as to yield seventeen to twenty-two grammes. The urates throughout were abundant, but their exact amount was not determined.

The records of this case of pneumonia have been thus fully given, in the hope that it may serve as a model for the investigation and recording of similar cases. Till such inquiries are fully carried out by many observers, working independently and uniformly, the statistics of pneumonia will get more confused than they now are, and will therefore become even of still less value.

It is now beginning to be appreciated by thoughtful physicians that the so-called physical signs are inferior in value to those afforded by accurate observation of the correlations of temperature, pulse, condition of the urine, and tendency to crisis; and these observations, so ably and clearly recorded by Dr. Parkes, are in unison with many similar records by Wunderlich, Metzger, Barthez, and others. They show that the crisis may occur on any day of the disease, but the usual dates are the *fourth,* or *sixth,* or *seventh* day.

The following is a tabular statement of those results:—

TABLE.

Date.	Day of Disease.	Condition of Patient.	Amount of Urine in cub. cents. in twenty-four hours. (*b.*)	Urea in grammes in twenty-four hours. (*a.*)	Chloride of Sodium in twenty-four hours, in grammes. (*a.*)	One kilogramme of body-weight excreted in twenty-four hours of urea, in grammes.
Jan. 10.	Part of sixth and seventh days.	First day of defervescence, temp. 100·6° Fahr.	980	85·56 (= 1,321 grains).	—	1·345
„ 11.	Part of seventh and eighth.	Temp. 98·6° Fahr. Complete defervescence.	—	—	—	—
„ 12.	Part of eighth and ninth.	Complete defervescence. Absorption of exudation.	865	87·38 (*b.*) (= 1,349 grains).	—	1·373
„ 13.	Part of ninth and tenth.	do.	865	87·38 (= 1,349 grains).	—	1·373
„ 14.	Part of tenth and eleventh.	do.	—	—	Some amount unknown. 0·040 grms. in 10 c. c.	—
„ 17.	Part of thirteenth and fourteenth.	Complete convalescence. Exudation gone.	1300	35·1 (= 542 grains).	5·2 (= 80 grains).	0·551
„ 24.	Part of twentieth and twenty-first.	do.	1297	30·2 (= 466 grains).	17·18 (= 265 grains).	0·474
„ 25.	Part of twenty-first and twenty-second.	do.	1760	44·0 (= 679 grains).	21·95 (= 339 grains).	0·691
„ 27.	Part of twenty-third & twenty-fourth.	do.	1910	40·1 (= 619 grains).	14·32 (= 221 grains).	0·630
Mean excretion of urea by one kilogramme of body-weight on the seventh, ninth, and tenth days during resolution, ...						1·363
Ditto ditto during the fourteenth, twenty-first, twenty-second, and twenty-fourth days' complete convalescence, ...						0·586
Mean physiological excretion in men aged twenty-two, ...						0·500

(*a.*) The urea and chloride of sodium were determined according to the method of Liebig, by Dr. Parkes' excellent Clinical Clerk, Mr. Smith. The chloride of sodium was not got rid of before testing for urea, but the usual correction was made. The amounts are given both in grammes and grains.

(*b.*) The urine of two days added together, and the total halved.

The Urine in Pneumonia.—(1.) The amount of *water* is lessened by a third or a half, (600 to 800 c. c.) This is most marked during the early days, and is no doubt due to retention of water in the febrile body. During the stage of resolution the amount of water increases. (2.) *The urea* is greatly increased during the height of the disease—an increase not due to any other cause than the metamorphoses of tissue during the pyrexia; and in equal periods of time it is more abundant during the day than during the night; and is in larger quantity *before* than during resolution. Dr. Parkes, however, has found it to be very great during resolution; and the general fact must be admitted that *the*

greatest amount of urea coincides with the highest temperature, and that it arises from general increased metamorphosis. (3.) *The specific gravity* is high (1025-1035), partly from deficiency of water, and partly from excess of urea; and as the chloride of sodium is absent, the specific gravity measures pretty accurately the amount of urea. (4.) *The uric acid* is increased, and its free excretion is a favourable sign. (5.) *The urine-pigment* is increased two or threefold, or altered; and it tints the urates, when they fall, brown, red, or carmine. (6.) *The chloride of sodium* is diminished, or is entirely absent during the existence of the fever and the occurrence of the lung-lesion—*i. e.*, during the early period, or at the commencement of hepatization. Even when given by the mouth at this period, it will not pass off as in health, but is retained, and will not appear in the urine (in Dr. Parkes' case, for more than forty-eight hours). It re-appears during, or rather after resolution, but it does not always re-appear directly resolution commences. It may still be retained for some days—as many as *eight* or *ten*—after which it is poured out in such quantities as sometimes to raise the specific gravity of the urine. This especially occurs in cases where there is little expectoration and no purging, although the water of the urine is increasing and the urea is diminishing. *Fifteen* to *twenty-five grammes* may be thus passed in the urine during twenty-four hours; and the period of its increase is some time after the excretion of urea has reached its acme, and is declining. There is thus ample evidence that the chlorine has been retained, and not excreted in excess through the skin; and even when *hydrochloric acid* is given to the patient, no chlorine appears in the urine; and Beale's observations prove that the lung-exudation is very rich in chlorides, which are often largely excreted in the sputa. The retention of the chlorides thus seems to be intimately connected with the development of the lung-lesion or exudation; and their excretion is increased during the absorption and disappearance of the exudation. (7.) *The sulphuric acid* is also increased by about a third. (8.) *The phosphoric acid* and the free acidity are lessened.

Abnormal products abound in the urine. They are chiefly as follow:—(1.) *Bile-acids;* (2.) Bile-pigment, with or without jaundice; (3.) Albumen is present in a large proportion of cases. The period of its occurrence is variable; but both Weller and

Parkes agree that it is most common at the commencement of and before resolution—*i. e.*, at the height of the disease. It has been supposed that some of the absorbed exudation is got rid of in this way (J. W. BEGBIE); but in the cases observed by Dr. Parkes, the albumen in the urine always appeared *before* resolution, when the lungs were yet commencing to be consolidated; and in one case there were, at intervals of from *six* to *eight* weeks, three recurrent attacks of acute pneumonic consolidation, affecting different parts of the two lungs, and at the height of each attack albuminuria occurred; while in the intervals, and subsequently, the urine was quite free. Dr. Parkes, therefore, does not ascribe the appearance of albumen in the urine in cases of pneumonia to the absorbed exudation passing out by the urine in this form, but to the implication of the kidneys in the general congestion and exudation, the most marked local seat of which was in the lungs.

Of five cases in which albuminuria occurred during the height of the disease, no fewer than three died; while among seven *non-albuminous* cases observed by Dr. Parkes, only one died. While the mortality of all the cases recorded by him was *four* in *thirteen*, or 30·7 *per cent.*, that of the cases with albuminous urine was 50 *per cent.*, and with non-albuminous urine it was 14 *per cent.*; "*therefore albuminuria occurring before resolution appears to be an unfavourable sign;* and the very frequent occurrence of albuminuria," continues Dr. Parkes, "in sthenic pneumonia is one of the most interesting facts in its clinical history. In no disease seen in this country, except, perhaps, in *maculated typhus*, is albuminuria so common as in *pneumonia*. *Bronchitis* of the large tubes, chronic phthisis, and emphysema present a most marked contrast to pneumonia in this respect." It is not invariably connected with *extent* of local disease: cases of enormous consolidation, and with great dyspnœa, have not necessarily albuminous urine.

In some cases, instead of being increased, the urinary solids are lessened, even in intensely febrile cases, with a heightened temperature of *five or six degrees*. Such is a result of retention, and not of diminished excretion (see vol. i., p. 56, *et seq.*); and in such cases some one or other of the following events happen:—(1.) At a later period of the disease a large amount of some ingredient may be poured out by the urine, although the febrile

symptoms have almost or entirely gone—*e. g.*, uric acid—the crisis by the urine of the older physicians; (2.) Towards convalescence spontaneous diarrhœa may come on; (3.) The recoveries are not so rapid as in cases in which the urinary excretion is large. The amount of the urine, indeed, is a good guide for prognosis. As in *typhoid fever* and other acute affections, it is probable that the products of metamorphosis are retained, and poison the blood.

During convalescence the urine generally augments considerably in amount, and (cases of previous retention of excreta being excluded) the *urea*, the *uric* and *sulphuric* acids diminish for a short period below their healthy range; the *chloride of sodium* increases, and an extraordinary amount is sometimes passed; but if the quantity got rid of by *sputa* or by *diarrhœa* has been large, the amount of *chloride of sodium* excreted by the urine will be proportionally less (Parkes *On the Urine*, pp. 270-279).

Decrease of temperature, as a rule, precedes the falling of the pulse, and, on an average, the *fourth day* is the day of *maximum* elevation in uncomplicated cases; the decline, beginning on the following day, becoming rapid, and attended with various well-marked critical phenomena on the *seventh day;* while the *minimum* is reached about the *ninth day.* In typical cases, throughout the attack, the temperature rises all day towards evening, and falls during the night towards the morning.

If a fresh lung-lesion or exudation commences, a re-accession of temperature will indicate its occurrence; but in all pure and simple cases the defervescence is a precipitate one, the temperature falling, in the course of from *twelve* to *thirty-six hours*, as much as 7° Fahr., or more degrees. In the majority of cases the decrease during the first *night* of defervescence is very considerable, small during the succeeding day, but again considerable during the following night (Wunderlich). If a diagram of the temperature in a case of pneumonia be projected upon paper by the student, as was done with several of the febrile diseases in vol. i. (some of which it resembles, such as *measles*), he may perhaps better appreciate the statements that have been made regarding temperature in this disease.

The natural history of pneumonia would be incomplete without some attempt to expound the causes of this tendency to a spontaneous favourable termination of uncomplicated cases, and their determinate duration. This cannot be better expressed than in

the words of Dr. Parkes. First of all he considers the following question:—

"What is the exact connection between the lung-symptoms and the general pyrexia? The course of the two is certainly not exactly parallel. The complete consolidation seems to be posterior, in point of time, to the height of the pyrexia. It has even been supposed by Wunderlich that the exudation into the lungs coincides with the end of the pyrexia—that is to say, that the defervescence commences when the lungs become completely hepatized. I have not been able to satisfy myself rigorously on this point. If it could be satisfactorily made out, it would certainly imply that the exudation into the air-cells relieved or cured the fever—in other words, that the lung disease is not a primary, but a secondary condition, and that it succeeds to and brings to an end, by purifying the blood, a condition of general pyrexia, arising from blood disease. Without believing that this relation is quite determined (if it were determined, the case would be settled), there is no doubt that the fever ends spontaneously, or very greatly lessens at the time when the inflammation of the lung is very great.

"There are at present two views more or less clearly defined which aim at explaining these phenomena, and which may be thus expressed—

"1. According to the first of these, there is a blood disease of some sort, of a nature not thoroughly known, but which consists, in part, in an augmentation of the fibrin, as in acute rheumatism. Increasing up to a certain point, and giving rise to the slight malaise which precedes all cases of pneumonia, it is at last brought to a head by some exposure, by a dietetic error, or by reaching a point at which the functions of the blood are seriously interfered with. Then ensues high general fever, from implication of the nervous system, and at the same time some organ or other is, on account of special affinity for the morbid blood, or from previous damage to its structure, specially irritated. In pneumonia the lung is the seat of election, and there is rapid hyperæmia and transudation of fluid into the air-cells. By this transudation the morbid blood is purified. The process is analogous to that of gout, in which a diseased blood gives rise to a local disease by the deposition of urate of soda in and about joints. When the localization and consequent purification is finished, then the fever ends. There remains the lung-exudation, which gradually softens down, is partly expectorated, partly absorbed; and, in the process of absorption, it may produce again secondary contamination of the blood, and certain affections of other organs, which constitute those secondary affections which sometimes complicate the after-course of pneumonia.

"The weak points in this hypothesis are the want of definite indications

of the blood disease, and of its mode of production. There is some evidence on these points, but it is certainly not very great.

"Its strong points are the explanation it gives of the previous malaise; of the sudden outburst of fever, when the diseased blood implicates at last the nervous system; of the singular and rapid termination of the pyrexia at a time when the lung-lesion is yet intense; and of the enormous elimination of urea during the very first days before the lung-exudation has softened down.

"2. The second hypothesis is the exact contrary of the former. The lung affection is supposed to be the primary lesion: it is a local inflammation produced by the (still obscure) causes of local inflammation, running the ordinary course of such inflammations, and giving rise to violent symptomatic pyrexia. The undoubted increase of fibrin in the blood is supposed to be not primary but secondary, to be caused by and to augment with the inflammation, and to be at its height with it. Virchow has stated that this fibrin is nothing more than the albuminous substance absorbed by the lymphatics of the inflamed and hepatized lung, and poured into the blood. The fever is believed to be entirely symptomatic of the local disease, and to be commensurate with its intensity and extent. The arguments for this view seem to be, that the lung-symptoms are remarkably early in manifestation, though they may not be very intense. Pain in the side and cough are very soon present, and sometimes occur even before the shivering and headache. The pyrexia, although great in the early days, is perhaps not greater than might have been produced by the condition of the lung; and as to the termination of the fever, this may be supposed to occur because the really true febrile stage of pneumonia is not the period of complete exudation, but the preceding period of intense hyperæmia. To say that the pyrexia is gone, when the lung-lesion is yet most intense, may be an incorrect expression of the fact: the consolidation may possibly, indeed, be most intense, but this may be merely the natural termination of that enormous hyperæmia and blockage of vessels from local changes of nutrition, which is in reality the essential disease.

"The difference between these two hypotheses would be this: the fever ends spontaneously, first, because the blood is purified, or, secondly, because the local disease ends spontaneously—*i. e.*, the active febrile-making local disease. This last assumption, however, is decidedly a very bold and hazardous one.

"Between these two views it is not very easy, nor perhaps is it desirable, yet to choose, for the blood has not yet been sufficiently examined. The only blood disease which has yet been indicated by the supporters of the first view, as anterior to pneumonia, is hyperinosis (excess of fibrin); and as hyperinosis occurs in acute rheumatism without

pneumonia, it is evident that there must be some other cause, either in the blood or in the local structure of the lung, which locates the disease in that part.

"That hyperinosis is really anterior in pneumonia as in rheumatism must, in spite of the opinion of Virchow, be considered likely, from the experiments, among others, of Professor Naumann, of Bonn: that it is not the only condition in either of these cases will be generally admitted. But what other blood affection is there? None has yet been indicated, to my knowledge, in the acute sthenic pneumonia of young persons without gouty or renal disease. But there is one point on which I have been trying to collect evidence for some years, but at present without sufficient success. It is well known how frequently the liver is affected in pneumonia, so that some amount of jaundice is not at all uncommon, and sometimes bile-pigment appears in the pneumonic sputa. I have also found in some cases evidence of liver affection for some time before the lung disease, especially the so-called torpor with deficient biliary flow.

"Is there, then, any condition of the liver which adds something to the blood which ought not to be there? Taurin has been found in the healthy tissue of the lung; but in the hepatized lung it seems, from Verdeil's observations on his pneumic acid (taurin), to be in excess. Is it some compound of this sort which, in combination with the hyperinosis, determines the localization of the blood disease, or produces by its irritation the inflammation of the lung? I know no facts whatever which can lead to a decision; but it is to be hoped that some competent person will soon undertake a more complete analysis of blood in the very first days of pneumonia than has yet been made.

"But, whatever be the facts as to the order of things in pneumonia, whether the lung affection is a mere localization of the blood disease, or whether the undoubted blood disease of the developed stage is merely produced by absorption from the inflamed lung, it is certain that the usual course of pneumonia is such as we witnessed in this untreated case, viz.,—(1.) There is an early period of intense fever, ceasing, if no complication be present, of itself, at a tolerably determinate time; (2.) There is a later period of lung hepatization, which softens down during a period of moderate fever, and is expectorated or absorbed. There are therefore two periods in pneumonia, and both have their dangers. The intense early fever may kill by its intensity; the exudation may kill, subsequently, by apnœa, or may contaminate the blood during softening to such an extent as to lead to renewal or increase of the fever and inflammation of other parts, or to coagulation of the blood in the heart or great vessels. Each period has its own dangers, and must have its own treatment."

Morbid Anatomy.—Following Andral and Laennec, all subsequent

writers on pneumonia are persuaded that a knowledge of the anatomical characters of the lung in this disease is essential to a perfect appreciation of the symptoms, physical signs, prognosis, and treatment of the disease. Both in the acute and chronic forms of diffuse inflammation of the substance of the lungs, the vesicular pulmonary tissue is more loaded with dark venous blood than usual, and its texture is more easily broken down than in health; air, however, still penetrates the air-cells, and consequently the lung still crepitates, swims in water, and, if washed, the colour is nearly restored, but it is doughy, pits on pressure, and is red or livid, and heavier than the normal lung.

This is the *first stage of pneumonia—namely, that of simple engorgement.* This state of inflammation may terminate by resolution, or it may pass to more complicated and dangerous lesions. Dr. Stokes is of opinion, however, that a *stage of irritation* has previously existed, so that the *first* stage described by Laennec would be the *second* described by Dr. Stokes, who has repeatedly seen a condition of the lung, regarded by him as the first or earliest stage of pneumonia, in which the lung-tissue is drier than usual, and of a bright vermilion colour from intense arterial injection; but there is no engorgement (*On Diseases of the Chest*, p. 310). Skoda and Fuller are opposed to this conjectural view.

When effusion of serum succeeds, the lung is in the same gorged state, but in addition it is loaded with watery fluid, so that on cutting into it the serous fluid mixed with blood streams from it as from a sponge. When its action is listened to during life, a lung in this condition no longer crepitates, and its bulk is enlarged; for it may be seen after death to have taken the impression of the ribs, and it does not collapse when the chest is opened. The lung is now technically said to be in a state of *red hepatization*, or, as Andral has termed it, *red softening;* for, although firm, its texture has lost its natural toughness, cohesion, and resistant power.

This is the so-called *second stage of pneumonia.* The diseased part readily breaks up by a thrust of the finger into its substance. This state has many degrees, the air-cells being either wholly or partially impermeable. In some instances the productive effects of inflammation (commonly called exudation) are very large in quantity, mixed with blood, and the more fluid portion can be readily separated, or, as it were, pressed out of the lung. In the

other extreme of this form of inflammation the exudation forms an integral part of the lung, which then becomes so solid that, if cut, it represents with much accuracy a portion of the liver or spleen. In this state it contains at the diseased part little or no air, does not crepitate nor float in water; it cannot be injected, is of a deep venous colour, while its texture is easily broken down and penetrated by the finger. The lung is still enlarged or swollen, and does not collapse when the chest is opened. It varies in colour from a reddish brown or deep dull red to a violet hue, and is generally darker in the aged than in the adult.

Suppuration may follow this form of pneumonia, and the pus effused may be either infiltrated or limited, as if contained in an abscess. Infiltration is by far the most common; and although this form of lesion may occur suddenly, as a result of serous infiltration in unhealthy persons, or during the progress of general diseases, yet, in the belief of most pathologists, it more generally follows the *red hepatization*. In this latter case the pulmonary tissue, red, dense, compact, and impermeable to air, passes to a grey colour, and hence it is termed *grey hepatization* or *grey softening*, and is regarded as the *third stage of the disease*, following Andral and Laennec. The structure in other respects of either form of hepatization appears to be the same; for if the lung be examined with a microscope the same granulations (only they are white or grey, instead of red) are found. There are instances, however, in which these granulations are wanting, and then a grey smooth surface only is observed. Hence the *granular* and *non-granular* forms of hepatization described by some authors. In aged persons the granulations of a pneumonic lung are much larger than in the adult, depending, no doubt, on the increased size of the air-cells in persons of advanced life (MM. HOURMANN, DECHAMBRE, and DR. MACLACHLAN). Hepatization in old people, with granulation, is nearly four times more common than non-granular or planiform hepatization (MACLACHLAN, l. c., p. 279).

In the *grey*, as in the *red hepatization*, the pulmonary tissue is easily torn, and the quantity of pus infiltrated is sometimes so great that, on cutting into the lung, that fluid readily flows from it: at other times the pus will not flow on a simple incision, but exudes by compression. The lung is solid and impermeable to

air. It sinks in water, and has ceased to crepitate. The interspersion of red and dark points gives a granite-like appearance to the section of a lung which is in a state of grey hepatization. The lung is then more friable than in red hepatization. The finger sinks into the grey portions of the lung upon the least pressure, and when the grey texture is squeezed it breaks down into a pulp.

Abscess of the Lung.—Although pus is more commonly diffused through the pulmonary parenchyma, yet sometimes it is collected into an abscess. In the infancy of pathology physicians regarded phlegmonous abscess of the lung as a common and ordinary occurrence, but it is extremely rare; and Laennec, when he published the first edition of his work, had only met with six cases, notwithstanding his extensive research; and in the practice of other physicians, phlegmonous abscess of the lung is equally uncommon. Abscess of the lung (although termed phlegmonous, to distinguish it from tubercular abscess) generally exists without any great intensity of inflammation, or other considerable alteration of its tissue. In the experience of Dr. Maclachlan, however, the rarified condition of the lung in the aged "seems to favour the formation of small abscesses, which are occasionally seen interspersed through the red and grey consolidated tissues, as if certain air-cells had broken down and coalesced during the plastic or suppurative process, and formed so many sacs for the reception of the effused or secreted matter" (l. c., p. 279).

Gangrene of the Lung.—Pneumonia may terminate by gangrene, which is also as rare a termination as by abscess. It occasionally arises from excess of inflammation, but more commonly the inflammation which precedes this state is of little intensity, so that it rather approaches to anthrax, and may be of pestilential origin. The gangrenous portion may or may not be circumscribed, and it is found in the different states of gangrenous eschar, of deliquescence, of sphacelus, and lastly, of simple excavation, the dead portion having been detached and expectorated. (See "Gangrene of the Lung.")

Pyæmic Inflammation and Abscess.—In some classes of cases, as in the continued fever of typhus and typhoid, pneumonia occurs as a local lesion, and is sometimes named "typhoid pneumonia;" so also in another class of cases, characterized by *blood-poisoning* or by *embolism*, the lung disease is a secondary result,

and is said to be due to pyæmia. While in yet another class of cases, as in those where some morbid condition of the constitution prevails—as in the alcoholism of drunkards—a primary pneumonia in them may give rise to blood-poisoning and the phenomena of pyæmia, by the sanious decay of the exudation and its re-absorption during resolution, or its passage to the left side of the heart.

Pneumonia may be either single or double—that is, it may attack one or both lungs at the same time. Of the part of the lung attacked, inflammation of the inferior lobe is most frequent; next, of the superior lobe; while rarely the whole lung is inflamed.

Bronchitis may take place without pneumonia, but in many cases pneumonia follows as a consequence. Pneumonia also may take place without bronchitis, but in general bronchitis accompanies it. Pneumonia also may take place without pleuritis, but it generally happens that the pleura is more or less affected. It is so especially in the pneumonia of pyæmia. In old persons primary pneumonia is mostly lobar, and the disease should be particularly looked for at the inferior and posterior parts of the chest. It runs its course very rapidly to purulent infiltration, and the *prodromata* are not distinctly marked. Parotitis is a frequent complication, and of bad omen, and the pneumonia may be marked by cerebral phenomena.

Hypostatic Pneumonia is that form which is apt to occur in those who are obliged to lie on their back for a length of time, on account of a fracture, paralysis, or the like.

Much speculation has been entertained with respect to the more particular seat of pneumonia; some contending that the inflammation affects the connective-tissue of the lung, others the air-cells, and others both. It is quite certain, however, that the minute bronchial tubes are not affected in slight pneumonia; for in such cases their divided extremities stand out in the midst of the inflamed part like so many white points. When the lung is more acutely inflamed, the bronchial tubes are red, and evidently greatly inflamed. There can be no doubt, however, from what we now know of the nature of the inflammatory process, and also from direct observation upon the tissue of inflamed lungs, that the minute elements of the vesicular and connecting tissue composing the parenchyma of the lung are from the first directly altered in their vital properties. (See vol. i., p. 64, *et seq.*)

General Symptoms of Pneumonia.—Pneumonia is generally preceded by some antecedent fever, by shivering more or less violent, and often by bronchitis. In a few cases, however, consolidation may be complete ere any primary affection is observed. In others, failure of the appetite, general weakness, and wandering pains in the limbs and chest, precede any definite attack by several days.

The disease being set up, the patient is restless and uneasy; his respiration difficult and hurried—from 30 to 50 in a minute—according to the amount of lung whose function is suspended; his cough frequent, and his expectoration streaked with blood; but notwithstanding this symptom he seldom, unless the pleura is affected, suffers pain, which consequently increases the danger. The aged, however, seldom complain of difficulty of breathing during the progress of the disease, whatever may be the frequency of the respiration, so that it is incumbent to count the movements of the chest in old people, to avoid all sources of error (HOURMANN, DECHAMBRE, and MACLACHLAN). Obscure and latent forms of attack, in the experience of these eminent physicians, are frequently met with in aged subjects labouring under chronic disease of the brain, heart, or some other internal organ. The pulse is full and frequent—from 100 to 120; the countenance livid; the nostrils dilated; the tongue and lips more or less livid, the former of which is coated with a white or yellow mucus. The patient inclines to lie on his back, supported by pillows. If he recovers, these symptoms are gradually mitigated; but should his case tend to a fatal end, the tongue becomes brown and typhoid, his pulse more rapid, profuse sweats break out all over his body, and at length his mind wanders, and he dies by *coma* or *apnœa*. There are many instances, however, where the course is widely different, and in which the patient, though evidently distressed by impeded respiration, has yet moments of cheerfulness; gets up, and may even walk about. But he suddenly dies, seized with a severe paroxysm of dyspnœa or of coughing, followed by collapse of the remaining healthy part of the lung.

In typical cases of acute pneumonia certain groups of symptoms are more or less constantly combined,—namely, accelerated respiration, with more or less difficulty of breathing, and rapidity of pulse. These are so correlated that the severity of the case is best measured by observing the correlation of these symptoms. The cough is at first dry, but soon there appears a scanty, semi-

fluid, grey, frothy, mucous expectoration, which becomes more and more viscid, and which, in the first stage of the disease, remains of a catarrhal yellow or white appearance. Afterwards, during the second stage, it becomes still more tenacious, reddish, or rust-coloured; and the heat of skin increases, with thirst and marked prostration, and a pulse increasing in rapidity.

Such are the general symptoms of pneumonia; and, except by their different degrees of intensity, it is difficult, if not impossible, to distinguish the different stages of inflammation from each other, without the aids of percussion and auscultation. The general symptoms of serous pneumonia, however, are the most marked; the uneasiness being greater, the respiration louder and more difficult, the countenance more livid and swollen, the cough more harassing, the expectoration more abundant, and the attempt to lie down impossible. A gangrenous state of the lung is determined chiefly by the intolerable fetor of the breath.

The duration of a case of pneumonia varies greatly. Laennec conceived that the diffuse inflammation lasted seven or eight days. We have seen that in simple, uncomplicated cases the crisis is reached about the fourth or sixth days (WUNDERLICH, PARKES, METZGER). Andral records the duration of the disease in the adult at eleven days; Chomel and Laennec, at from seven to twenty days; Bouillaud, from eight to twelve days; Hourmann and Dechambre, fourteen days. The mean duration in the aged, of 109 carefully recorded cases by Hourmann and Dechambre, was found to be nine days and seven-tenths; and Dr. Maclachlan agrees with them in considering the progress of pneumonia to be more rapid in the aged than in the adult. More generally, however, taking all its forms, cases of pneumonia terminate between the seventh and the twentieth day.

Physical Signs of Pneumonia are to be appreciated by *inspection, palpation, mensuration, percussion,* and *auscultation. The physical signs of pneumonia,* in the adult and in the aged, are so dependent on the morbid changes which have been described in the lung in the various stages of the disease, that a description of them is best given in correlation with the morbid anatomy of the disease. In doing so, the descriptions given by Dr. Fuller in relation to adults, and by Dr. Maclachlan in relation to the aged, will be mainly adhered to.

On striking the chest of a person in health it returns a certain

hollow resonant sound, demonstrating it to be partly filled with air. Also, if we place the ear to the chest, we hear certain sounds, on inspiration and on expiration, which are termed the respiratory or vesicular murmur (as described at page 541, *ante*). In pneumonia these natural sounds are altered, the sound on percussion being rendered duller than natural, while the bronchial respiration undergoes still more remarkable alterations; and these modifications enable us, by percussion and auscultation, to determine the nature and seat of the disease. During the *first stage*, inspection of the chest shows that the costal movements are not materially diminished, unless the motions of respiration are restrained by pain; and it is only by percussion and auscultation combined that the existence of pneumonia can be determined.

By *percussion* a peculiar euphonic resonance may be emitted by that portion of the chest at which pneumonia is beginning, which ceases after the commencement of exudation, to be replaced by dulness more or less marked according to the extent of the lung involved (FULLER). During the period of invasion in the aged, the chest continues to sound clear over the seat of the impending inflammation; and as congestion increases, the lung becoming more dense and less permeable, the sound emitted on percussion may be somewhat duller; but in general there is no appreciable alteration till the *second stage* has set in (MACLACHLAN).

The signs elicited by *percussion* depend on the consolidation of the tissues, the amount and position of the consolidated part in relation to the surface of the chest, and the amount of intervening healthy lung. If the consolidation is near the surface, the sound is decidedly dull, and the sense of resistance to the extremities of the percussing fingers is considerable in proportion to the amount of the consolidation. To emit this unequivocal dull sound of hepatization in middle age, the disease must be extensive, and without intervening healthy lung; but in some cases, if the hepatization be central, the air in the more superficial portions of healthy lung often prevents a dull sound from being returned on percussion. In such cases percussion elicits a clear but shallow resonance. A slight diminution in the resonance of the chest, immediately over the affected portion of the lung, is usually all that percussion elicits in old age, in the second stage of pneumonia. By auscultation during the second stage, tubular breathing is found to be a

most constant accompaniment alike in the adult and in old age. The respiration is of a hollow character, diffused throughout the hepatized portion—tubular towards the centre, but harsh and blowing towards the periphery of the affected part. Where the tubular breathing prevails, râles and rhonchi are absent; while vocal resonance is intense, usually of a metallic ringing character. This is most marked in aged persons. The *ægophony* in them is more decided, the shrill, acute, tremulous voice of old age being more favourable to *ægophony* than to *bronchophony*. When fibrinous exudation is great and complete, blocking up vesicles and tubes completely, absolute dulness on percussion, with little or no vocal resonance, can alone be heard; and the heart's sounds, greatly intensified, are then frequently transmitted through the consolidated lung.

Auscultation during the early period of engorgement, before exudation has taken place, finds the breathing weak in the affected parts, and exaggerated in their immediate vicinity. In the aged, tubular breathing is sometimes audible at the root of the lung at a very early period, if the respiration is weak. But as soon as fluid exudes into the affected parts, the respiratory sounds are obscured or replaced by the small crepitation characteristic of pneumonia. This sound, called crepitation, is made up "of a multitude of minute crackles, which occur in a volley towards the end of inspiration, and are unaffected by coughing or expectoration" (FULLER). It is the true crepitating râle of Laennec, and has been variously compared "to the crackling of salt thrown upon the fire," or to "the sound produced by rubbing a lock of hair between the finger and thumb close to one's own ear" (WILLIAMS). It usually, but not invariably, accompanies the accession of pneumonia; and it is very seldom present in advanced life. In subjects beyond the age of fifty, and particularly in still more advanced age—as between sixty and eighty—the bubbles composing the pneumonic crepitation are very generally larger, more hurried, and less numerous. The râle essentially resembles the subcrepitation of capillary bronchitis, and is very often speedily marked by copious accumulation in the larger bronchi (MACLACHLAN, GRISOLLE, CAZNEUVE, HOURMANN, DECHAMBRE). When the air-cells are completely filled by the exudation, and the minute bronchial ramifications obstructed, crepitation ceases; and when capillary bronchitis commences,

small bubbling râles are heard accompanying both expiration and inspiration. When acute pneumonia arises in connection with *acute rheumatism*, Dr. Fuller has often observed that crepitation never occurs. This, he believes, is attributable to the occurrence of the exudation into the interlobular connective-tissue, and consequent exclusion of the air-cells (l. c., p. 221). Vocal resonance is generally intensified. The return of the respiratory murmur, and the gradual disappearance of crepitation, indicate the resolution of the disease—a process which is generally more tedious and less perfect in the aged than in the adult—often followed in elderly persons by symptoms of continued irritation of the lung or bronchi. On the other hand, in proportion as crepitation marks the respiratory murmur, and replaces the pulmonary expansion, the supervention of consolidation may be anticipated.

Beyond the limits of dulness and consolidation, the extension of fine crepitation denotes the extension of the *hepatization;* but when resolution has commenced, crepitation again returns to the part which was dull and consolidated before; or moderate sized râles are heard, denoting the passage of air to and fro amongst the fluid which occupies the air-cells and smaller bronchial ramifications; and very soon the percussion note assumes its normal character.

During the *second stage*, inspection of the thorax shows the costal movements diminished in the affected side, and they may be somewhat increased on the unaffected side; but the movement of elevation is less affected than that of expansion.

By *palpation* we learn that *vocal fremitus* is usually above the average: and Dr. Fuller has verified the observation by *post-mortem* results, which showed that when the bronchi were plugged with exudation, vocal fremitus was entirely abrogated. The *fremitus* produced by a deep-toned powerful voice is seldom observed in the aged, and even *bronchophony* is occasionally absent (Maclachlan).

The passage from the first to the second stage of pneumonia is generally extremely rapid in old age, sometimes within a few hours (Maclachlan). In the *third stage* of the disease the physical signs do not differ from those already described; and neither in the adult nor in the aged are any certain phenomena appreciable by which the passage from the second to the third stage can be determined. A persistence of the sounds already

described, in place of those denoting resolution, and especially when the expression of the phenomena diminishes with increasing prostration of the patient, and diminished energy of voice and respiration, betokens suppuration of the lung. It is by the succession of the physical signs in the adult and in the aged; by the occurrence of a rhonchus composed of large humid bubbles, without crepitation, in a portion or portions of lung previously consolidated; by the coincidence of this state with increased exhaustion; by a peculiar dusky, cachectic expression of the patient; by feebleness of the pulse; and by typhoid symptoms and bloody seropurulent sputa,—that no room is left for doubting the accession of suppuration of the affected part (MACLACHLAN).

Fine crepitation of the lung cannot always be regarded as pathognomonic of acute pneumonia. In some instances it is absent throughout the attack, alike in the adult and in the aged. "It is only," as Dr. Fuller justly observes, "by the concurrence of different signs that it is possible to arrive at a trustworthy conclusion. Fine crepitation occurring coincidently with intense heat of skin and rusty coloured expectoration, warrants the strongest suspicion of pneumonia, and justifies the adoption of active treatment; and if it be accompanied by marked alteration in the ratio of the pulse and respiration, speedily followed by dulness on percussion and tubular breathing, the existence of acute pneumonia cannot be doubted. But crepitation, however fine, if not attended by an alteration in the ratio naturally subsisting between the pulse and the respiration [and I would add, the temperature], and not speedily followed by tubular breathing, cannot be relied upon as indicative of pneumonia. If it occurs without these symptoms, it is commonly indicative of capillary bronchitis with scanty secretion; whereas, if, under the same conditions, it is accompanied by dulness on percussion, it is probably due to rapid œdema, or else to congestion of the lungs, connected with some febrile hæmic disorder, cardiac disease, or the deposit of tubercle" (l. c., p. 229).

Pneumonia, it has been stated, sometimes, though rarely, terminates in abscess. The physical symptoms previously to the bursting of the abscess are those of hepatization; but supposing the abscess to have burst into the bronchial tubes, the pus of course escapes, and a cavity filled with air is left, communicating with the bronchial tubes, and this new state of parts gives rise to

a new series of phenomena. The air, for instance, having penetrated into the cavity, the part which returned a dull sound while the abscess was yet unbroken, will now return a sharper and clearer sound on percussion than natural, denoting a larger admission of air than takes place in health. Again, on auscultating the chest, we find some changes have taken place both in respiration and in the transmission of the voice. If the cavity, for example, be large, and the opening small, the natural respiratory murmur at that part will be superseded by a sound resembling a person blowing into a jug, and from this circumstance termed by Laennec, "râle amphorique," or "bottle sound." Again, if the cavity be large and its walls dense, and the abscess still contains some pus, we hear a sound as if a drop of water had fallen into a pool; and this sound is so sharp and metallic that it has still preserved the designation originally given to it by Laennec, of *metallic tinkling*. It is usually supposed that this sound is produced by a globule of pus or fluid dropping from above into the fluid below; but some are inclined to believe that it is owing to the bursting of a bubble of air, mixed with the pus of the abscess. If, on the other hand, the abscess be large, and contains some pus, on the patient coughing we actually hear a *splashing sound* of the pus against the walls of the abscess, especially if the chest of the patient be quickly moved to and fro (succussion).

In aged persons, pneumonic abscesses are generally numerous, of limited extent, and interspersed through the lung. Physical diagnosis of them is therefore scarcely possible; but if the abscess be large, the sudden evacuation of purulent sputa, with gurgling and cavernous respiration, denotes the circumscribed suppuration of an abscess cavity.

Another circumstance revealed by auscultation, in the event of an abscess, is *pectoriloquy*. This physical sign is heard when, the stethoscope being applied to the chest, and the patient desired to talk, we hear his voice as if he were speaking directly at the end of the stethoscope, the sound passing to the ear as through an ear-trumpet.

Pectoriloquy, however, does not take place in all cases of abscess of the lung: its occurrence may be considered the exception rather than the rule in this disease. The cause of this is, that many conditions are necessary to its existence; first, that the lung

must be condensed so as to have some conducting power, or the voice will be destroyed, as in health, before it reaches the aperture communicating with the abscess. Again, it is necessary that the patient should have a sufficient quantity of voice to produce strong vibration; but this is often wanting. Another condition is, that the bronchial opening of the abscess be not too large, for in that case the vibrating force is diminished. It is likewise injurious to the effect that there should be not more than one opening into the abscess; for in that case not only is the vibrating force diminished, but the counter motions of sound destroy all vibration. It is plain, also, that the walls of the abscess must have a certain density, or their flaccidity will act as a damper, and destroy all vibration. Many conditions, therefore, are necessary to pectoriloquy; and we cannot feel surprised that one or more of these signs of an abscess cavity may be wanting.

Besides an opening into the bronchial tube, the abscess may at the same time open into the cavity of the chest, and this new pathological state gives rise to the sound of a *metallic tinkling* of the chest, infinitely more powerful than that caused by a simple pulmonary abscess. Indeed, the intensity and sharpness of the sound quite equals that returned by a copper vessel when struck with a slight force; for the intercostal muscles brace the walls of the chest like a drum, so that they become an excellent conductor of sound. The immediate cause of the sound is supposed to be exactly the same as when it results from an abscess; that is, either a drop of fluid falling into the pus below, or the extrication of a bubble of air from the gravitated pus. The chest in these cases always returns a remarkably clear sound on percussion beyond the precincts of the fluid.

Diagnosis.—Pneumonia is distinguished from phthisis by the previous good health of the patient, and by the more acute nature of the disease; and, in some degree, by a difference of its seat, the lower lobes being more particularly affected in inflammation, the upper lobes in phthisis. The two diseases, however, it should be remembered, are often combined. In determining distinctly the further diagnosis of pneumonia, from the phenomena of condensation and respiratory murmurs, it is necessary to bear in mind the various conditions which may produce condensation of the lung either in the child or in the adult. The observations of Laennec, Legendre, and Baillie,

Jörg, Fuchs, Barthez, Rilliet, West, and Gairdner, have especially elucidated this subject, and shown its importance as an element to be attended to in diagnosis. The following is a short statement of the conditions which lead to consolidation of the vesicular tissue of the lung:—(1.) A partial unexpanded state of the lung in a new-born child, termed *atelectasis* (JÖRG). (2.) Consolidation connected with the accumulation of mucus in the bronchi and air-cells—*bronchitis* in the infant, followed by what has been called *lobular pneumonia* of children, the *état fœtal* of the French (FUCHS, WEST, ZENKER). (3.) Collapse of the pulmonary air-cells, causing *lobular* or *more diffuse* forms of pulmonary condensation in adults, as well as in children, due to bronchial obstruction (GAIRDNER). (4.) It is probable that the *hypostatic pneumonia* described by Piorry, and the *péripneumonie des agonisans* of Laennec, and *some* of the so-called *latent pneumonias*, are forms of condensation due to *pulmonary collapse*, combined with serous effusion or vascular congestion. (5.) The consolidation of inflammatory lymph in or about the pulmonary vesicles, so that the vesicular tissue of the lung is imbedded in solid material, "as the stones of a wall are in mortar." (6.) Condensation of the lung from the pressure of pleuritic effusion. (7.) Condensation of the lung from extravasation of blood (apoplexy of the lung), or from tubercular or cancerous deposits; or enlarged bronchial lymphatic glands, which are arranged along the sides of the air-tubes in their passage through the substance of the lungs.

Seeing, therefore, that consolidation may result under such a variety of conditions, the truth of the statement so well expressed by Dr. Stokes must at once appear in the strongest possible light —namely, "That in the cases we are every day called to treat, the value of physical signs must be tested by the history and symptoms (records of temperature and the like), and these in their turn must be corrected by the physical signs." In truth, no disease shows more forcibly than cases of pneumonia do, and especially as regards diagnosis and treatment, that *every individual case of disease requires to be made a special study as regards its individual history, progress, combination, and sequence of symptoms.* In the aged, pneumonia is extremely dangerous, and often difficult to detect.

Treatment of pneumonia has been, and seems with some still to be, one of the most discordant topics in the Science of Medicine.

At one time, especially about fifty years ago, large bleedings appear to have been demanded, and to have been well borne. Laennec and Louis seem to have been early impressed, from their experience, that large bleedings were by no means an eminently successful practice, and that in some cases they were absolutely injurious. The same difference of opinion was held with respect to large doses of the *tartrate of antimony*. These discrepancies are now to be explained by the circumstance that pneumonia, like other inflammations, not only varies in type and severity in different cases, but that "extremes in practice," whatever they may be, when adopted as a *rule absolute*, applied to all cases, can never give satisfactory results. (See also p. 725, *ante*.)

The ancients bled in pneumonia, and sometimes to deliquium, and Galen appears to have adopted this practice. This was also the practice of Sydenham; and Laennec says it was common in France at the beginning of the last century to take twenty-four, thirty, and thirty-six ounces of blood at one bleeding. This practice was also followed throughout Europe at that time, and there can be no question of its having been proper in cases of simple inflammations, like pneumonia, occurring in healthy persons. It is clear that the remedy was used with a moderation apportioned to the severity of the disease and the strength of the constitution; or it was withheld altogether when the patient was weak, or the disease assumed a low type.

Now, however, it would be an unwarrantable error to make excessive bleeding the basis of remedial measures in all cases (see vol. i., p. 157, *et seq.*); but there can be no doubt that we have it in our power materially to modify the course and to shorten the duration of pneumonia by the judicious employment of blood-letting, leeches, tartar emetic, certain salines, and opium (PARKES).

In epidemic pneumonia, says Laennec, it is hardly possible to bleed the patient without rendering him worse. In 1814 pneumonia was very common among the conscripts, yet there were few indications for bleeding, and those that were bled were rendered much worse. With respect to bleeding in cases of pneumonia, much must be left to the discretion of the practitioner. That there are cases in which the patient can only be saved by general blood-letting, everybody must admit, while on the contrary, when pneumonia is epidemic, the quantity of blood drawn

must be greatly limited and the case well watched. The ancients held that bleeding should not be practised after the fifth day, as it prevented *concoction.* In other words, it tended to change the type of the disease, in its natural tendency to a cure, so that the processes tending to the resolution of the inflammation by the natural *cell-therapeutics* of the part were altered for the worse. The cases of Louis appear to establish the propriety of early bleeding as a general rule; for he says those bled in the first four days of the affection are cured four or five days sooner than those who are bled later in the disease. The experience of Dr. Alison is similar. In short, as a general rule, the earlier the inflammatory state is detected (if possible, before the third day—ALISON) the more likely will bleeding be followed by well-marked beneficial results—the disease will be sooner cured, and the convalescence more rapid and perfect.

It is rare now that the cure of pneumonia is left entirely to the influence of bleeding. Antimony is a most valuable remedy; and we owe to Rasori the introduction, in modern times, of large doses of *tartar emetic* in the treatment of pneumonia. Laennec was so dissatisfied with his own results of bleeding that he adopted it, and thus describes his practice:—

"As soon as the disease is determined, if the patient be in a state to bear bleeding, I take from eight to sixteen ounces of blood from the arm. I do this as momentarily arresting the inflammation, and thus giving the tartar emetic time to act, and I rarely repeat this bleeding. Immediately after it I give the first dose of tartar emetic, or a grain in two ounces and a half of orange-flower water, and I repeat this dose every two hours for six times: I then allow the patient to repose for six or seven hours. If, however, the disease be severe and the oppression great, I continue the dose every two hours till the symptoms are mitigated, increasing the dose from one to two grains, or even to two grains and a half." The immediate effects of this practice were, that the larger number of patients vomited two or three times, and had five or six stools on the first day, but afterwards the evacuations were trifling, and when tolerance was established, they often required purgative medicines; while many bore these large doses almost without vomiting or experiencing any purgative effect. The result was that Laennec is said to have cured 27 cases out of 28 in 1824 and in 1826. The great success

obtained by Laennec appears, however, to have been of short continuance, for M. Lagarde afterwards published an account of 16 cases treated by Laennec by this method, of whom 5 died. Lecoultreux has given a list of 30 cases likewise treated by Laennec, and of whom 12 died. Neither have other physicians in other years been more fortunate. Louis treated 15 cases according to this method, and 6 died; Chomel, 140 cases, and 40 died; and Gueneau de Mussy treated 90 cases, of whom 38 died. Andral has treated a considerable number of cases of pneumonia by tartar emetic, in quantities varying from six to thirty-two grains in the twenty-four hours; and he adds,—"I have seen but two unpleasant or grave accidents result from these large doses. Sometimes the patient has not been at all affected, and has neither had nausea, vomiting, diarrhœa, or abdominal pains; at others he has suffered from nausea and distressing vomiting—effects which have subsided on omitting the medicine. Tartar emetic," he adds, "may therefore be given with impunity. But is it useful? I have not seen pneumonia ameliorated by large doses of this medicine; for neither has it appeared to do good when borne by the stomach, nor when it has excited distressing nausea and vomiting."

Great doubts are now entertained regarding the usefulness of such large doses of antimony. The Rasorian method is decidedly contra-indicated in the pneumonia of old people; but when the remedy is cautiously administered as a sedative, or diaphoretic and expectorant, in doses of *the eighth* or *twelfth of a grain* every *two* or *three* hours, and in doses of a *sixth* or a *quarter of a grain*, in the more sthenic attacks, it is a most valuable remedy (MACLACHLAN). It is contra-indicated in gastric or gastro-enteric irritation. There are two kinds of cases where the remedy produces severe depression and nausea—cases in which the stomach will retain *no* food, scarcely even water when given alone. It is under such circumstances that Dr. Anstie, with justice, advocates the administration of alcohol in stimulant doses often repeated. He cites one case of double pneumonia in a man aged twenty-four, who, beginning with twelve ounces of brandy *per diem*, ultimately took twenty-four ounces in divided doses every half hour, and for *ten* days lived on *nothing else*, and a little water. In one month he was able to resume his work. A child fourteen months old, under similar morbid conditions, was similarly treated

with port wine and water. Taking about six ounces of wine a day, in spoonful doses, with water, the child subsisted on the wine and water *alone* for twelve days. At the end of that time, the stomach still refusing food, a little cod-liver oil was substituted for the wine, and in ten days more the appetite for ordinary food began to show itself, and the child made a good recovery (*Stimulants and Narcotics*, by Dr. Anstie, p. 446).

But in the statistics which bear upon the treatment of pneumonia, little or no reliance can be placed as a guide to practice. The results revealed are so variable and contradictory as to deprive them of the slightest claim to authority. For example: —*Without depletion*, Dr. Bennett's statistics show a mortality of 1 in 21·4; Dietl's, 1 in 13; the homœopathic treatment (mainly expectant), 1 in 6; Vienna, 1856, 1 in 4. *With antimony and bleeding*, Grisolle lost 1 in 8; Dr. Bell, Glasgow, 1 in 17·7; Trousseau, 1 in 26; Wossildo, 9 in 76. *Treatment by the inhalation of chloroform*, Baumgartner, 1 in 10; Varrentrapp, 1 in 23.

Former statistics in the Royal Infirmary of Edinburgh are said to show a mortality of one in three.

These results are clearly too variable a guide to any sound basis of practice. No satisfactory conclusion can be drawn from cases massed together. Each case must be studied by itself, as well as each epidemic. (See also pp. 725 and 747, *ante*.)

The numerous qualifying conditions connected with age, season, climate, epidemic and endemic influences, earliness of treatment, stage, extent, and complications, all tend to modify the line of treatment required. Dr. Lawson, Professor of Medicine in Cincinnati College, has well expressed these conditions, in a general sense, under the designation of "*the individuality*" of each case.

"Indeed," he remarks, "so great are the differences in constitutions, that no two examples will exhibit the same characteristics throughout, nor will they admit of precisely the same method of treatment. It is a due appreciation of these more minor shades of differences, as well as the broad distinctions observed in the varying *forms* of the disease, that denotes the truly skilful physician, and which enables him to meet the emergencies of each case, instead of relying on conclusions drawn from groups of cases. It has been remarked that English physicians are apt to think more of some other case they have seen than the one under treatment; while the French think more of the disease than of the patient.

Hence English physicians *individualize* the disease; the French physicians *generalize* the patient.

"The true course has been indicated by Hufeland—namely, *to generalize the disease and individualize the patient.* The tendency of statistics is undoubtedly to rob each case of its individuality. Thus, one group of cases will all be bled, another will receive tartar emetic, a third will be left to the chances of Nature. In the first group some are bled who ought to have been stimulated; in the second group *tartar emetic* is administered when *bleeding* would have been preferable; in the third group some are permitted to die from over-action. Thus the individuality of a case is ignored, and the physician prescribes for a mere *name.*"

A rational treatment is one which must secure to each case its own individuality. And as the shades of differences and the corresponding modifications of treatment cannot be expressed in *groups,* statistics in this sense become simply an impossibility. For example, bleeding, antimony, mercury, and blisters may be demanded in one case; quinine, opium, and wine in the next; a third may require but little interference beyond a well-regulated diet, with moderate stimulants.

The treatment of pneumonia, therefore, demands not a single, but many agents. It is the proper *combination* of remedies, not a single agent or mode of practice, which must be sought for in the treatment of pneumonia (LAWSON).

It now only remains to add the more matured opinions entertained as to the line of treatment to be adopted with pneumonia as it now occurs. In cases of sthenic pneumonia characterized by intense heat and dryness of the skin, a full resistant pulse, rusty coloured expectoration, *and great oppression of the breathing,* blood-letting, had recourse to at the beginning of the attack, in the stage of congestion before the *fastigium* of the pyrexia, not only affords immediate relief to the breathing, but appears to remove the extreme tension of the vascular system, and to promote secretion. The most suitable time is that during the evening exacerbation (HUSS). The indications for blood-letting should be decidedly marked before it is undertaken; and the amount of blood to be lost must vary in each case, according to the oppression of breathing, the type of the disorder, and the constitution of the patient, as well as to whether or not there is any prevalent epidemic tendency associated with the pneumonia. It is seldom necessary to draw

more than from *ten* to *sixteen* ounces; and *eight* or *ten* ounces will usually suffice. After bleeding, the pain in many instances ceases, expectoration takes place more easily, and alters in character; the skin becomes moister, and evidence is afforded of the action of remedies which before proved inoperative (FULLER). In very many cases it is now either unnecessary or inexpedient to let blood—inexpedient chiefly because of the constitution of the patient being shattered by excesses, constitutional disease, anxiety, and mental distress; and excessive blood-letting, under whatever circumstances practised, impairs the strength, leads to great impoverishment of the blood, arrests the actions on which the absorption of exudation-matter depends, exposes the patient to risk, and induces a tardy convalescence.

Bleeding, therefore, ought certainly not to be employed after consolidation has taken place.

A combination of *antimony* and *calomel* is believed to have saved a much larger number of cases than *antimony* alone; a quarter of a grain to a grain of the *tartrate of antimony,* combined with one grain of *calomel,* given every four or every six hours, according to the severity of the disease, is the treatment in some cases to be adopted. Previous to its use the bowels should be well cleared out, and after the mercurial effects are indicated by the condition of the gums, the further administration of the remedy should cease. In cases of simple serous pneumonia even simpler remedies are sufficient; and two grains of *ipecacuanha* given every four or six hours have frequently been followed by the recovery of the patient. The cases in which *tartar emetic* fails to exercise a curative action are those in which hepatization proceeds with extreme rapidity; in which crepitation either does not exist at all, or is of very short duration, giving place after a few hours to intense tubular breathing; and those which are marked by extreme depression almost from the first.

Salines and *stimulants,* with *calomel* and *opium* if necessary, are found to be the most efficient means of treatment in such cases. The efficacy of *mercury,* in the experience of Dr. Fuller, is most conspicuous in those cases of pneumonia in which *tartar emetic* is of least avail; in other words, in those instances in which the productive results of inflammation are the greatest, in cases in which crepitation does not exist at all, or is replaced in a few hours by intense tubular breathing (*On the Chest,* p. 238).

In these, *calomel* and *opium*, in combination with *salines* and small doses of *tartar emetic*, will often produce very beneficial results.

Alkaline Treatment of Pneumonia.—Dr. John Popham, Physician to the Cork North Infirmary, since 1865, has treated twenty-eight cases of pneumonia by alkalies. They all recovered. In some the signs of engorgement only existed at the time of admission; in others, hepatization was found. The reliable marks of pneumonia were present in all—*e. g.*, localized dulness, crepitation, tubular breathing, rusty sputa (in many), and labial herpes in about half. Six were cases of double pneumonia. *Bicarbonate* of potash was given, largely diluted in mucilaginous liquids, five grains up to thirty grains in each dose,—four, six, or even eight doses being given in the twenty-four hours. An adult took to the extent of two to three drachms of the salt per day. The evidence of its good effects appeared first in altering the character of the expectoration on the second or third day of its use. The viscid sputa became resolved; the fine bubbles became coarse and large; the rubiginous colour of the expectoration was changed to white; its tenacity of adhesion was lessened, so that it was brought up easily; and the cough, instead of continuing dry, harsh, and irritative, became moist, soft, and expulsive. Thus the alkali acted as a sedative, allaying the cough and abating the congestive state on which it depends. The white pasty fur over the tongue dissolved away in an increased flow of saliva. The urine became alkaline, and the physical signs of pneumonia became resolved. A blister applied for four or six hours (not for suppuration) is a valuable auxiliary; suppuration from blistering is exhaustive and prejudicial (*Brit. Med. Jour.*, Dec. 28, 1867, p. 586).

The treatment by repeated stimulants alone is but the opposite extreme of practice to excessive blood-letting, and will no doubt soon find its level, and appropriate place as a valuable aid to other remedies, especially in patients whose constitutions are depressed under the influence of life imposed upon them by *the "great town system."*

PHTHISIS.

LATIN EQ., *Phthisis;* FRENCH EQ., *Tuberculeux;* GERMAN EQ., *Phthisis* —SYN., *Schwindsucht;* ITALIAN EQ., *Tisi cronica polmonale.*

Definition.—*The growth or exudation of a peculiar material, in*

the form of a tubercle, which undergoes various changes in the lungs, associated with the constitutional phenomena of scrofula.

Pathology.—The nature and treatment of this disease, commonly known as *pulmonary consumption* or *phthisis pulmonalis*, have been already fully described, from p. 188 to p. 230, and it is only with reference to its diagnosis in the lungs by general symptoms and local physical signs that it requires notice here.

Tubercles may be developed in a slow and insidious manner:—(1.) In the form of the granulations described (p. 193, *et seq.*), and confined to one portion of the lung, where they gradually increase in size and number; or, (2.) They suddenly and rapidly increase, and are found in many parts of a uniform size, generally called miliary tubercles, which are regularly distributed through the pulmonary tissue. They present different stages of retrograde metamorphoses at different parts of the lung. The recent grey tubercles are soft and gelatinous, those of older date are firm; and when the tissues are extensively invaded, they are softened and infiltrated with a thick serous effusion. The course of such cases is generally acute, and hence "acute tuberculosis" as a form of scrofula. In acute phthisis they may reach the size of a pea in three or four weeks (Louis); and when subjects in a state of scrofulous cachexia are exposed to violent irritation of the lungs, these granulations grow so rapidly and in such numbers throughout the lungs as to give rise to most alarming dyspnœa (Sir James Clark). Tubercle also occurs—(3.) As an *infiltration of the tissues* by an albuminous fluid of a thick synovial-like character, which gradually degenerates into a firm greyish-red granular softened mass, containing portions of tissue within it. This infiltration was first described by Dr. Baillie in his *Morbid Anatomy*. "In cutting into the lungs," he writes, "a considerable portion of their structure sometimes appears to be changed into a whitish soft matter, somewhat intermediate between a solid and a fluid, like a scrofulous gland just beginning to suppurate. This appearance is, I believe, produced by scrofulous matter being deposited in the cellular [areolar or parenchymatous] substance of a certain portion of the lungs, and advancing towards suppuration. It seems to be the same matter with that of tubercle, but only diffused uniformly over a considerable portion of the lungs, while the tubercle is circumscribed." According to the pathology of the present day, as now described, this infiltration of *scrofulous* fluid *tuberculizes*, and the

tubercles form themselves in the first instance in the cavities of the air-vesicles.

It has been much discussed (and still is discussed), in which texture *tubercle*-matter is first deposited, whether fluid or solid, and what is the cause of the deposition or growth. The deposit in the lungs is the one most frequently described; and while we have had some most minute and elaborate descriptions in recent numbers of the *Medico-Chirurgical Review*, it appears they do no more than confirm the account which Schroeder Van der Kolk gave forty-two years ago (1826), when he fixed the seat of tubercular *deposit* in the extremities of the bronchial tubes, or in what are named the pulmonic air-vesicles. Similar views were adopted by Carswell and Andral. Dr. Sieveking has since confirmed the observation by his own researches; Schroeder Van der Kolk has again elaborately described the more minute details as to how the process takes place; and Dr. Radclyffe Hall gives a similarly minute description. One form of tubercle appears in the air-vesicles in the first instance as a degeneration of the previously existing normal epithelium; shedding off this, and continuous replacement and degeneration, till the whole of the interior of the air-vesicle is filled with tubercle. Subsequently the pulmonic fibres become enclosed and separated by the morbid growth and exudation, and free nuclei and granules are formed between and amongst them. According to Virchow, such epithelium becomes "cloudy," and the cells degenerate, and the process of tuberculization advances in the cavity of the air-vesicle. This is also consistent with the experience of Hasse, who is of opinion that the tuberculous matter is first deposited on the walls of the vesicles, and gradually presses towards their middle the epithelium-cells, or other contents of the air-vesicle, and then encloses the whole as a kind of soft and sometimes dark-coloured nucleus. Another fact is of interest in relation to this point. In a stone-mason who died of phthisis an earthy nucleus was found at the centre of many of the pulmonary tubercles. This earthy nucleus turned out to be of precisely the same character as the stone of Craigleith quarry, where the man had been employed (Home, *Edin. Med. and Surg. Jour.*, January, 1838).

The question of epithelium in air-cells is still the subject of discussion. Its existence is disputed by Rainy, Todd, and Bowman, Mandl, Ecker, Deichler, Zenker, Henle, and Munk;

but its existence is maintained by Kölliker, Radclyffe Hall, Williams, and Hasse. It is also asserted by Eberth that in the lungs of calves and pigs there is a very delicate unbroken epithelium (Virchow's *Archiv.*, 1862, p. 503, where figures are given). By preparing healthy human lungs from young subjects by boiling the lungs in dilute acetic acid, I believe I have succeeded in demonstrating the existence of a delicate film of pavement epithelium lining the air-cells.

With regard to the origin of pulmonary miliary tubercle, an important observation is made by Deichler, which would indicate the occasional origin of tubercle in another way. He found, when a section was made through the centre of one of the tubercle-masses, that it enclosed a little stem, knuckle, or portion of a minute pulmonary artery, about one-twentieth of a line in size. This knuckle, or loop of artery, was surrounded by tubercle. The walls soften by the new formation of cells; and the more the cells grow, the larger the tubercle-mass becomes, till it is finally lost in the larger knots. It is not improbable that the tubercle-knots are formed owing to a diseased state of the membranes of the stems of the blood-vessels. Evidence of such disease may be found in cases of *ectasis* or aneurismal distension of the walls of the pulmonary capillaries, as seen in the air-cells. Buhl and Zenker have described such cases (Virchow's *Archiv.*, 1862, p. 183). Bearing upon this point, I lately dissected a soldier who died suddenly by hæmorrhage from the lungs. On opening into one of the tubercle-cavities, it was found filled with coagulated blood; and projecting from a spot on the wall of this pulmonary cavity was a round tumor about the size of a walnut. This tumor had ruptured, and the rupture held a coagulum of blood. The tumor was found to be an *ectasis* or aneurismal dilatation of the pulmonary artery; and several other tumors of a similar nature, but of variable and much smaller sizes, existed in other cavities in the lungs, projecting from the pulmonary artery. They were proved to be continuous with this vessel—(1.) By the injection of spirits into them, and their distension thereby through the pulmonary artery as it left the heart; and (2.) By microscopic examination, which showed a delicate epithelial lining to these tumors, continuous and similar to that in the artery. The preparation is preserved in the museum of the Army Medical School at Netley. (See also *Lancet*, Feb. 6, 1841; and referred to in *Prov. Med. Association Trans.*, vol. x., p. 65.)

It may shortly be summed up, with regard to tubercle in the lungs (as Gluge long ago showed), that the ultimate pulmonic vesicles are often involved in scrofulous infiltration, giving rise to *one* form of isolated tubercle (*tubercula granulata*), confined, perhaps, to a dozen alveoli, the principal part of the morbid growth being in the alveoli.

Another form of isolated tubercle in the lungs occurs as an interstitial growth, situated in those points where a certain amount of connective tissue exists. Its occurrence is in the form of little knots, or separate grains massed together, but always in their early stage recognized as bright sparkling spots like sand (SIBSON),—the usual *miliary tubercle.*

The seat of tubercle in the lungs may therefore be the mucous membrane of the bronchial tubes entering the alveoli, the walls of the alveoli themselves, the connective-tissue of the lung, the walls of the pulmonary artery, or the surface of the pleura.

Cases are rare in which the occurrence of tubercle is not at first latent in the lung; and from the numerous cicatrices that are found after death at the apices of the lungs, it is exceedingly questionable whether any person is carried off by a first attack. Tuberculous disease of the lungs in early life frequently heals, although it generally returns (sometimes at an advanced age), and ultimately proves fatal (SIR JAMES CLARK). There are few cases, also, writes Dr. Bennett, in which the destructive and ulcerative progress of scrofulous inflammation is uniform. It is continually being checked, and for a time slumbers; and even in the worst specimens of tubercular lungs numerous cicatrices and evidences of attempts to heal may be recognized; but as one portion cicatrizes another becomes the seat of further tuberculous growth. Cicatrices of healed tubercles present different appearances, according as the cavities from which they were formed have been superficial or deep-seated. When superficial, the pleuræ are more or less adherent and thickened, frequently thus forming an external boundary to the cavity. When the walls of the cavern contract, the pleural surface of the lung is drawn inwards, and thus the irregular puckerings on its surface are produced. Occasionally no traces of scrofulous matter are discovered either within or in the vicinity of these cicatrices; but more generally the contraction and puckering of parenchyma occur round tubercle which has undergone various transformations, and sometimes a cyst encloses

the mass. The enclosed growth may be found to have undergone any of the processes of transformation already noticed (p. 193, *et seq.*); and the cretaceous or calcareous concretions may remain an indefinite time in the parenchymatous substance of the lungs, or they may be evacuated through the bronchi with the sputa. Thus masses of tubercle are sometimes absorbed or thrown off, and the evidence of this may be summed up under the following points:—(1.) Small puckered cicatrices with loss of substance, after subsidence of the systemic disturbance characteristic of pulmonary phthisis. Strumous growths in the lungs of children seem capable of absorption without undergoing any change such as cretification or softening. Most frequently it seems in them to affect mainly the small lymphatic glands along the outer walls of the air tubes. (2.) The conversion of tubercles into cretaceous or horny masses, which are either expectorated or remain latent. (3.) Isolation of the growth by so-called plastic material; adhesion and thickening of the pleura; adhesions and lymph thickenings round cavities, analogous to cysts round clots of blood or musket balls. That such appearances are really evidences of arrested tubercles is rendered apparent by the following facts:—

"1. A form of indurated and circumscribed tubercle is frequently met with gritty to the feel, which, on being dried, closely resembles cretaceous concretions.

"2. These concretions are found exactly in the same situations as tubercle—most commonly in the apex, and in both lungs. They frequently also occur in the bronchial, mesenteric, and other lymphatic glands, and in the psoas muscle, or other textures which have been the seat of tuberculous growths or scrofulous abscesses.

"3. When a lung is the seat of tubercular infiltration throughout, recent tubercle generally occupies the inferior portion, and older tubercle, and perhaps cancer, the superior, whilst cretaceous and calcareous concretions will be found at the apex.

"4. A comparison of the opposite lungs will frequently show that whilst on one side there is fine encysted tubercle, partly transformed into cretaceous matter, on the other the transformation is perfect, and has occasionally even passed into a calcareous substance of stony hardness.

"5. Seeing that, according to the observations of Dr. W. T. Gairdner, cicatrices also may result from bronchial abscesses, the seat of cicatrices in the lungs may vary considerably" (Bennett).

In the words of Carswell, "pathological anatomy has perhaps

never afforded more conclusive evidence in proof of the curability of a disease than it has in that of tubercular phthisis." Pathology, therefore, teaches most distinctly that the aim of the physician must be to correct the constitutional tendency to the further occurrence of tubercle, or even to arrest, if possible, at its origin, the development of the constitutional state which leads to such growths. The most eminent physicians are agreed that it is peculiarly a disorder of childhood and youth, "when nutrition is directed to building up the tissues of the body," that such persons are frequently attacked with symptoms of phthisis, which under proper treatment cease, "and years elapse before there is any renewal of the disease, and that were advantage taken of the intervening period to correct the constitutional cachexia, the cure might prove complete." During remissions of the systemic disease, there is toleration of the lesions; but the remission ceases with renewal and extension of the local lesions.

The result of the course followed by tuberculosis, when observed in patients under treatment in hospital, may be arranged, according to results obtained by Dr. Walshe, under the following heads:—

1. All the symptoms were removed and the physical signs reduced to a passive condition in about 4½ *per cent.* of the cases admitted, without reference to the stage of the disease, or to the severity of the primary or secondary morbid changes.

2. If the persistence of some active physical signs are disregarded, such as a continuance of cavernous rhonchus, all the general symptoms disappeared in nearly 8 *per cent.;* and complete removal of all the symptoms was more frequently effected in the *male* than in the *female.*

3. More than half the cases of phthisis undergo temporary stages of improvement more or less permanent; and the time the disease has existed, rather than the stage the disease has reached, is an important element in calculating the probable benefit a patient may derive from treatment in hospital.

"In a given mass of cases the chances of favourable influence from sojourn in the hospital (at Brompton) will be greater, in a certain (undetermined) ratio, as the duration of the disease previous to admission has been greater; in other words, natural tendency to a slow course is a more important element of success in the treatment of the disease than the fact of that treatment having been undertaken at an early period" (*Med.-Chir. Review,* Jan., 1849).

There are certain circumstances which promote the chances of a remission—namely, (1.) Originally good constitution; (2.) Non-inheritance of scrofula; (3.) Direct inheritance of longevity; (4.) Limited extent of local lesion; (5.) Integrity of other organs, unimpaired digestion, absence of fever, vigorous nervous system, quiet pulse; (6.) Influence of age—the period when the young body is making its growth is the period of greater danger. The tolerance of lesions and the periods of remission are less during the growing age, and are increased just when the body has completed its process of increase.

PROVISIONAL ARRANGEMENT OF THE VARIETIES OF CHRONIC PULMONARY PHTHISIS (DR. ANDREW CLARK).

Name.	Chief Anatomical Characters.
1. Tubercular, granular, or specific phthisis.	The true grey granulation. Pigmentary tubercle. Fibrous tubercle. Cellular tubercle?
2. Scrofulous or epithelial phthisis.	Primitive yellow tubercle; accumulation, cheesy degeneration, and disintegration of epithelium-like cells.
3. Catarrhal or bronchial phthisis.	Ulceration of bronchi, with adjacent fibroid and cellular deposits, and cheesy degeneration of the same.
4. Pneumonic phthisis.	Disintegration of recent or old deposits occurring in vesicular, lobular, or lobar pneumonia, primary or secondary, common or scrofulous.
5. Fibrous phthisis (cirrhosis, chronic, or interstitial pneumonia).	Fibroid deposits, with cheesy degeneration of imprisoned portions of lung, due to—(*a.*) Mechanical irritation (as in grinders, masons, miners, &c.); (*b.*) Rheumatic inflammation of interlobular tissue; (*c.*) Chronic pleurisy; (*d.*) Constitutional states, as in granular kidney and liver.
6. Amyloid phthisis.	Circumscribed or diffuse cellular formations infiltrated with amyloid material.
7. Syphilitic phthisis.	Cheesy disintegration of nodules of nucleo-fibrous tissue, and diffuse infiltrations of the same.
8. Hæmorrhagic phthisis.	Cheesy degeneration and disintegration of nodules of extravasated blood.
9. Embolic phthisis, including pyæmic deposits and suppurations.	Cheesy degeneration and disintegration of grey or yellow deposits, arising, directly or indirectly, from pulmonary emboli coming from the liver, lymphatics, or veins.

Symptoms of Tuberculosis of the Lungs, or of Phthisis.—These correspond with the intensity of the expression of the constitutional state; they vary also with the extent of the local growth or exudation in the lung, and with the condition in which the tubercles exist, and the changes they undergo. As a general rule, it may be stated that in the ordinary chronic form the presence of tubercular matter in the substance of the lungs, whether in its semi-transparent and crude, or in its softened state, does not cause much pain to the patient beyond dull, aching, "flying" pains about the collar-bones, or under one or both shoulder-blades, occasional soreness of the throat, and tightness across the upper part of the chest.

The greater number of cases of chronic pulmonary tuberculosis are indicated in the commencement with some slight cough of a dry hacking character, most frequently induced on rising in the morning and going to bed at night. The cough at first seems simply intended to clear the throat, the irritation being mainly referred to the pharynx, which is often red, rough, and coated with tenacious mucus. As the cough becomes more fully expressed a scanty expectoration occurs, of ropy or glairy mucus, hardly discoloured, or only slightly streaked or stained by a trace of blood. Dyspepsia, sick headache, biliousness, and loss of appetite prevail. The patient is feeble, easily fatigued, feels unequal to his usual work, has burning heat of the soles of the feet at night, and some perspiration in the morning; his nights are restless, so that he rises in the morning weary and unrefreshed by sleep. He is irritable, and often depressed in spirits, his appetite capricious, with dyspepsia—a most constant, important, and early symptom,—and he is convinced of a sensible loss of flesh. His muscles become flabby, the countenance pale, the conjunctiva becomes pearly-white, and the pupil of the eye dilated. These symptoms are accompanied by a permanently accelerated pulse, from 90 to 140. The cough after a time begins to recur at intervals during the day, and especially after the least exertion. He is now sensibly aware of being "short-winded," so that active exercise exhausts him, and he must rest at intervals if he walks fast or goes up a stair. The number of respirations per minute is now increased; the pulse accelerated, especially towards evening, and deficient in force. Febrile paroxysms are persistent, and of daily occurrence; and elevation of tempera-

ture, as measured by the thermometer, is a constant phenomenon capable of being appreciated for many weeks before physical signs become decided (DAVY, RINGER). There is also correspondence between the elevation of temperature and the activity of the growth of tubercles in the lungs; such that when it goes on rapidly the elevation of temperature is high, and *vice versa*. This elevation of temperature is persistent, varying from 103° to 104° Fahr., for several weeks, before diminished weight or physical signs indicate the undoubted presence of tubercle (RINGER). This stage or state of things may last a few weeks or a few months; and even the patient often revives, and seems to an unpractised eye, for a short time, to have recovered his good general health, were it not for a sense of weakness and undue exhaustion after such exercise as he has been daily accustomed to.

The disease, however, silently proceeds, and all the preceding symptoms are gradually but sensibly aggravated. A large amount of tubercle may have grown during the earlier stage; and after a time the intervening portions of lung become so congested that bronchial irritation, with intercurrent attacks of pneumonia, are of frequent occurrence. Rapid softening of the tubercles takes place in those in whom the constitutional disease is intense; and with the continued discharge of pus from the lungs, hectic fever becomes permanently established, and the sweat from the head and chest towards morning is often so profuse that the patient lies bathed in perspiration. The cough is more distressing, the sputa purulent, and the pulse more frequent, perhaps from 90 to 110. The emaciation, consequently, is now well marked and decided. The duration of this the *second stage* is very indefinite; a few weeks to many months may end in a fatal issue. During its progress, however, the disease occasionally intermits and becomes latent, so that often there is for a time a marked amendment, and the patient regains some strength—a most important interruption, to be sedulously taken advantage of, for the purpose of promoting the tendency to cure. But fresh tubercles are apt to grow during these intervals, so that the lung-tissue is still further encroached upon by the newly-formed growth, fresh local irritation is set up, all the general symptoms are aggravated, sickness and rejection of food are now often excited—the expectoration becoming more profuse, of a purulent character, and often streaked with blood.

The third and last stage of this eventful constitutional disease is that in which, after the tubercle has softened, a cavity forms. In this stage all the preceding symptoms attain their highest degree of intensity: the hectic is followed by cold clammy sweats, and great exhaustion towards noon and again at night; the appetite is lost; a colliquative diarrhœa may supervene from tuberculous ulceration of the intestines; the sputa are often pure pus, as from an abscess, but at length become little more than a rusty sanguineous mucus; the pulse rapidly increases to 110 or 150; the emaciation is excessive; the hair falls off; catamenia cease; pleurisy or pneumothorax may supervene; ulceration of the larynx; and, amidst this general wreck of material existence, the Mind is often firm, collected, and even hopeful to the last.

As soon as the cavity which forms is in free communication with the bronchi, the cough is often greatly relieved. The duration of this stage is generally shorter than the former; but still, notwithstanding the existence of one or more cavities, it often lasts many months. As the fatal end approaches, the appetite fails more completely; sleep can only be obtained by narcotics; the integuments on which the patient lies are apt to become inflamed, sore, and even to die from the constant pressure; œdema of the feet and ankles sets in; and with the approach of death, suffocative dyspnœa may render the death-struggle extremely painful. In other cases a wandering delirium enshrouds the Mind, or coma supervenes, and the prolonged and weary illness is tranquilly ended by the gentle and welcome approach of death.

Such is a short outline of the course and phenomena of this destructive disease. It sometimes terminates life within a few weeks, or extends over six or eight months, while it occasionally lasts several years, with marked intermissions in its progress.

The following is a short analysis of the principal local, constitutional, and stethoscopic signs of this remarkable affection:—

Affections of the bronchial membrane and mucous membranes of the pharynx and larynx are certainly the most frequent concomitant symptoms of phthisis; but the part of the bronchial membrane affected is not always the same. Most commonly the mucous membrane of the smaller bronchial tubes is first affected; then that of the larger ones, the disease gradually ascending till it often ends in a *chronic laryngitis*, with a partial or total loss of voice. In a few cases, however, this order is inverted, and almost

the first symptom is a *laryngitis*, with *hoarseness*, partial loss of voice, and constriction of the throat; after which the disease descends to the larger and then to the smaller bronchi, when the patient begins to expectorate; his pulse becomes hurried; he loses flesh; and all the unerring symptoms of phthisis are established; while *pleurisy, pneumothorax, pneumonia, bronchitis,* and *hæmoptysis* are the more important intercurrent complications; while amongst the more chronic are *ulcerations of the bowels, scrofulous inflammation of the epiglottis, larynx, trachea, bronchial glands, or tuberculous meningitis.*

The *expectoration* which takes place in phthisis from the bronchial membrane is usually purulent, the pus thrown up in the early stages being for the most part of good quality, and formed into "sputa," sometimes sinking, and sometimes swimming in water. It may be either of a sweet, insipid, or saltish taste, as experienced by the patient. As the disease advances, pus is often expectorated pure, as from an abscess, and without any separation into sputa, and is sometimes mixed with particles of a curdy substance.

In the last stages of consumption it is often of a rusty green, a dirty sanies, or a rusty muciform serosity. The quantity expectorated varies greatly; sometimes only a few sputa, or not more than half an ounce in the twenty-four hours, and then perhaps more than a pint in the same period, so that in a few weeks the patient has often expectorated more than his own weight of pus. If a small abscess has burst into the bronchi, the sputa, though somewhat increased in quantity, are hardly changed in character; but if the abscess be large, the quantity thrown up is proportionally great.

Hæmoptysis.—Hæmorrhage may precede, or be contemporaneous with, or succeed to the bronchial affection. If it precedes, the patient being, as he imagines, in excellent health, is suddenly seized with *hæmoptysis*, followed perhaps by cough. This attack subsides, but a second and third follow, till the condition of pulmonary tuberculosis becomes undoubted. In this respect, however, the hæmoptysis of phthisis must be distinguished from the recurrent and minute hæmoptysis of an aneurism opening into the air-passages. Hæmoptysis more commonly, however, occurs later in pulmonary phthisis, increasing the debility, aggravating the symptoms, and hastening the fatal issue. The quantity of blood lost

is sometimes only enough to streak the sputa, at others a few tea-spoonfuls, but in some instances so profuse as to amount to one, two, or more pints. In the still more advanced stages, though cases occur in which the quantity of blood thrown up is very great, yet more usually it is trifling, and more resembles a bloody sanies than pure blood.

The *dyspnœa* is generally great in phthisis, the patient being unable to make any active exertion, or even to read a few lines without pausing. The dyspnœa, however, is not always proportioned to the amount of mischief; for there are instances in which the respiration has been performed with facility, even when two-thirds of the lungs have been in a state of tuberculoma. It is doubtful whether adhesions, unless very extensive, greatly affect the respiration. Should effusion of serum, however, or of pus, from the bursting of an abscess, have taken place into the cavity of the chest, then the respiration is greatly impaired. The most common situation of the fistulous opening, caused by the bursting of an abscess into the chest, is the summit of the lung, or a little below the clavicle. It is usually very small, hidden by the lung, or so surrounded by adhesions that it is difficult to discover it.

The Stomach is more or less diseased in three-fifths of the cases of phthisis; yet it so seldom gives rise to any well-marked symptom that for the most part any lesion there may be said to be latent. In the worst cases the symptoms are only a capricious appetite, indigestion, some pain in the epigastrium, and vomiting after coughing. The indigestion is peculiar, as already mentioned at pp. 207, 208. With reference to fat and fat meat, the dislike of consumptive patients to such food is unquestionable; so that the fat and the lean parts have to be adjusted to the likings of the patients, and it is generally of no use giving to them any but the leanest parts of the meat. They are partial also to fish, and especially to soles.

The Intestinal Canal is at least as frequently affected as the stomach in phthisis; but in general the abdomen is without pain. The only marked circumstance is, that the stools are more copious than in health, the body being unable to appropriate the accustomed quantity of nutriment prepared by the stomach. As the disease advances, the patient often suffers from irritable bowels, or from diarrhœa alternating with constipation; while, towards the close of the disease, the diarrhœa often becomes colliquative,

hastening the fatal result. In some few instances the peritoneum ruptures, and the patient dies of *peritonitis;* while in a somewhat larger number *ascites* is a common occurrence.

The Liver undergoes fatty degeneration in about one-third of the cases, and so remarkable a lesion might be expected to give rise to some particular symptoms; but this is not the case. It may occasionally be felt somewhat enlarged, but neither pain, nor altered state of the secretions, or other circumstance, denotes its diseased condition.

The emaciation so remarkable in this disease is common to nearly all the tissues of the body, as the adipose tissue, the muscles, the bones, and even the intestines and skin are thinned. This emaciation often commences before the disease can be said to be well established, so that the patient has often lost one or two stones in weight before he applies for medical advice. In the more advanced stages emaciation progresses in a peculiar manner, the patient losing perhaps three pounds in one week, and gaining two pounds or more in the next; and this alternation of gain and loss goes on for many weeks or months, but generally leaving a balance against the patient. Towards the close of life the loss greatly surpasses the gain, and occasionally amounts to four, five, six, and seven pounds in a week. The total loss the patient sustains is perhaps from one-third to half his whole weight.

The Mind, though not capable of continued exertion, is generally perfect throughout the disease, or only wanders during the few last days of existence. It is seldom the patient dreads the future or despairs of the present, for nature, however threatening his symptoms, has imparted a singular buoyancy to his hopes, and he always says he is better; would be quite well but for his cough; feels able to take a long walk; and sometimes (in expectation merely) enjoys his meals;—yet, with all this, he may faint if he attempts to cross the room, or nauseate when his food is brought to him.

In the aged, it has been shown by Dr. Maclachlan (*op. cit.*, p. 333), that "consumption may exist independent of tubercular development, and that tubercles are not the essential anatomical character of senile phthisis; and that the most extensive destruction of the lungs not unfrequently occurs, accompanied with the usual symptoms of this disease, without a trace of

tubercle." It is usually a sequence of chronic bronchitis, terminating in indolent inflammation and partial induration of the lung. These indurated parts at last break down, leaving caverns and burrowing sinuses, which are characterized by a dark and sloughy aspect of their inner surface, and by the absence of any membranous lining (ARMSTRONG, GRAVES, STOKES, MACLACHLAN). "It constitutes the *ulcerous phthisis* of Bayle, the *pneumonic phthisis* of Addison;" and, in the experience of Dr. Maclachlan, it "is far from unfrequent in aged persons, the victims of intemperance or of chronic visceral disease." He believes it to be a truly scrofulous form of inflammation, disorganizing the lung. In *senile tubercular consumption* the tubercular growths are generally confined to the lungs—commonly limited to one lung only; and to the upper and back portions—tubercles existing in the very apex or apices only, the remainder of the lung being healthy (MACLACHLAN, *op. cit.*, p. 334).

Such are the general symptoms of phthisis. A physical examination of the chest will yield many interesting additions to our means of diagnosis, and enable us to determine, not only that the lung is diseased, but also the particular part, and the state in which it may be. Thus the discoveries of Laennec and Avenbrugger have rendered the diagnosis of *tuberculosis* of these organs almost as perfect as though the disease was exposed to sight. Nevertheless, there is no *single sign* by which the existence of tubercles in the lungs is clearly indicated; therefore the general symptoms must invariably be judged of in combination and comparison with the information obtained by physical signs.

Not a little has been written as to the possibility of detecting what has been called a "pretubercular stage of phthisis" (E. SMITH and others). By this expression writers mean that there exists an abnormal physical condition of the lungs and of the body preceding the deposition of tuberculous matter in the lungs, which condition is capable of demonstration by certain signs or symptoms. Some physicians find that no local physical sign of disease is indicative of the existence of phthisis *before the growth of tubercle;* but the tendency to the tuberculous state is inferred from the presence of a series of cachectic symptoms, which from experience are frequently found to terminate in tuberculous disease of the lungs. Others believe not only that such symptoms

are present, but that they can detect such physical signs as are believed to indicate a peculiar local condition of the lung—*a stage anterior to the deposition of tubercle.* "The slightest subclavicular dull percussion sound, with lessened vesicular murmur, less forcible and deep inspiration, and flattening of the apex of the lungs," are described as the physical signs of this so-called *pretubercular* state (MARKHAM). But, however much practitioners may accustom themselves to detect the physical signs of known pathological conditions, there are states of the body between cachexia and local lesions so nicely balanced that no definite local morbid state can be ascribed to them, and therefore no physical signs can be associated with such a negative position apart from those of health. The so-called "pretubercular stage of phthisis is undoubtedly an instance of this kind. The best auscultators have admitted that there are no distinct and infallible signs by which we can with certainty diagnose the early existence of tubercle; and it is also known that solitary growths of tubercle do not of themselves produce the slightest change in the percussion sound of the lungs." On the contrary, most physicians believe that "the slightest subclavicular dull percussion sound, the less forcible, deep, harsh, or tubular inspiration, with lessened vesicular murmur, prolonged expiration, increased vocal resonance, and flattening of the apex of the lungs, when combined with the well-known general symptoms, leave little doubt that the actual presence of tubercle is indicated." While, therefore, it is imperative on the student and practitioner to educate their ear to the utmost in the detection and appreciation of the finest thoracic murmurs, it is unjust, as already observed, to expect through the stethoscope more information than it is fitted to convey. A too exclusive study of physical signs, to the almost disregard of general symptoms, not only does injustice to the science of Medicine, but the lives of patients are endangered when treatment is solely founded on the former. It is by general symptoms, such as those which Todd, Clark, Bennett, Ancell, Hutchinson, and Ringer have so fully elucidated, that the practitioner will be able to recognize a "pretubercular stage of phthisis," and not by local physical phenomena alone. A very interesting illustration of this is afforded by some recent observations made by Dr. Sidney Ringer on the value of thermometric observation as indicative of a pretubercular stage of phthisis. He has been able to recog-

nize a persistent elevation of temperature as the invariable precursor of the growth of tubercle in any organ. This persistent elevation of temperature existed for several weeks before diminished weight or other physical signs indicating the growth of tubercle in the lungs could be appreciated. After a certain time, however, growth of tubercle began to be apparent by physical signs in the apices of the lungs. His observations were made on patients in University College Hospital; and he kindly permitted me to read, in MS., a summary of his observations. (See also vol. i., p. 42.)

Physical Signs.—In the following details a systematic examination of the patient is prescribed, after the method recommended by Dr. Fuller, otherwise the diagnosis is a matter of extreme uncertainty to those unpractised in conducting a physical examination of the chest. The physical signs are obtained by—(1.) *Inspection;* (2.) *Palpation;* (3.) *Mensuration;* (4.) *Percussion;* (5.) *Auscultation* of the chest.

The physical signs of pulmonary tubercle, in the order of their relative frequency, may be stated as follows:—*Dulness of percussion* (constant); *deficiency of respiratory murmur; bronchial voice; rough inspiration; expiration prolonged; mobility of chest-wall lessened; flattening of chest-wall; tubular quality of respiration; dry crepitation; wavy inspiratory sound.*

If the chest of a patient labouring under incipient phthisis be carefully examined, we may at first observe nothing remarkable unless the growth of tubercles shall be large in amount or confined to one side of the chest. Abnormally rapid respiratory movements indicate a large amount of tubercle; and there is decreased expansion of the chest in the *infra* and *supra*-clavicular regions on the affected side, or flattening of the chest-walls at the place affected; and if both sides are equally affected, this flattening is apt to escape attention. If the disease be further advanced, we find the patient emaciated, together with a singular immobility or incapacity of dilatation of that portion of the chest immediately below the clavicle, so that he breathes chiefly by his shoulders and diaphragm, and is unable to "fill his chest," especially during forced inspiration. The respirations are now greatly increased in frequency. In the latter stages of the disease the respiratory movements are still more rapid. The clavicles appear peculiarly prominent, in consequence of the flattening, depression,

or falling in of the *supra* and *infra*-clavicular regions. A fluctuating impulsive movement may be obvious in the upper intercostal spaces, generally on the left of the sternum, due to the action of the pulmonary artery or base of the heart. At a still more advanced stage, if an abscess has burst into the cavity of the pleura, and caused *pneumothorax*, the affected side is not only motionless, but distended, and as it were bulging out.

Palpation during the *early stage* may disclose deficient expansion in the *infra* or *supra*-clavicular regions; and if there is increased vocal fremitus at the apex of the lung, it betokens consolidation of the lung-tissue underneath. In the *advanced stages* of the disease, marked vocal fremitus betokens a large cavity, superficial, and in free communication with the air-passages. It may be rhonchial, or gurgling, or like fluctuation. If the lung has shrunk, the heart may be felt beating out of its proper place.

Mensuration with calipers, in the early stage, may detect a slight diminution in the *antero-posterior diameter* of the *infra-clavicular* region, and a decrease in the local expansion movement; as the disease advances, the size of the side affected diminishes both in its *transverse* and *antero-posterior* diameter, probably due to atrophy and cicatricial-like contraction of the lung, collapse of air-cells, or firm pleural adhesions.

Percussion during the earlier stages, so long as air penetrates the pulmonary tissue equally on both sides of the chest, does not convey any definite information. Dulness does not always follow even when considerable masses of tubercle exist at the apex, unless the masses are very superficial, when the sense of resistance to the fingers is also increased. But if a portion of healthy or emphysematous lung intervenes between the consolidated lung and the chest-walls, there will still be clear resonance over the affected part. Percussion should be practised during deep inspiration, and then during deep expiration. If tubercles exist, it will then be found that in percussion under a full inspiration, the resonance is *increased* on the affected side, but very *slightly* as compared with that on the healthy side; whereas, if percussion be made under a full expiration, the dulness will be far greater on the affected side than over the healthy lung (FULLER, pp. 42, 44, 365). It is differences rather than actual quality of sound on which an opinion must be based. In the more advanced stages

percussion elicits evidence of dulness more intense and more widely spread. In the latter stages, where *vomicæ* exist, percussion will be absolutely dull if one or more small vomicæ are filled by purulent material, or surrounded by consolidated lung. If, however, the same cavities are empty and superficial, without thickening of the pleural membrane, the dulness may be slight, or the sound on percussion resonant, though shallow or amphoric. "Large empty vomicæ, with thin tense walls, yield an amphoric or cracked-pot resonance, . . . and, except in rare instances of enormous superficial vomicæ, with thin tense walls, it is almost impossible to judge of the size of a cavity by the results of percussion" (FULLER, *op. cit.*, p. 369).

A superficial empty cavity, with resilient walls, having free communication with the upper air-passages, yields a cracked-pot sound, especially when the mouth is open, and all obstacle to the egress of air removed.

Auscultation yields extremely variable results. Slight harshness of respiration is the first indication noticeable, with prolongation of the *expiratory* murmur, and "jerking irregularity of the respiratory sounds." When these phenomena are persistent, and confined to one side of the chest, they indicate tubercle, especially if the phenomena of "dry clicking" be added. These phenomena are earliest marked in the *supra-scapular*, *supra-clavicular*, and *infra-clavicular* regions. If the physical signs of bronchitis (namely, coarseness of respiration, with small bubbling râles and sonorous sibilant rhonchi) are persistent in these regions, and inaudible below the second intercostal space, and still more so when confined to one lung, their existence warrants the suspicion of tubercle. If the disease is advanced, and the growth of tubercle involve bronchial tubes of considerable size, there is almost entire absence of vocal resonance and of respiratory murmur over the part affected, with the sounds of breathing exaggerated in the adjoining parts. If consolidation is extensive, the sounds of the heart and large vessels are transmitted to a greater extent than in health.

The diagnostic value of murmur in the pulmonary artery is variously estimated. It is often present in many phthisical patients at the second left sterno-costal articulation (FULLER, p. 366. See also Sieveking, in *Lancet*, Feb. 11, 1860).

As the disease advances into the second stage, auscultation

indicates the presence of thin irregular-sized bubbling râles, from the passage of the inspired air through the softened and liquefied tubercles. It may also prove that coarse and hollow-sounding respiration exists over a more extensive surface than heretofore; or that the respiratory sounds are of a blowing character (bronchial breathing). In still more advanced conditions, with more or less empty cavities, the respiration is heard to be blowing or amphoric and metallic; and if the fluid contents of the cavity rise above the level of the permeable bronchi which lead into the cavity, large irregular bubbling râles or distinct gurgling may be heard. Well-marked pectoriloquy (from a mere whisper) is the form of vocal resonance most pathognomonic of pulmonary tuberculosis (FULLER, l. c., pp. 363 to 370).

The physical signs denoting a few small scattered tubercles are,—(a.) *Inspiration rough and dry, and its intensity increased;* (b.) *Expiration prolonged—compared with its normal intensity and duration, as eight to two;* (c.) *Bronchophony in rare cases* (POLLOCK).

Of crude tubercle in groups or masses the physical signs are,—*Pulmonary crumpling sounds; dry crackling rhonchus; sonorous sibilant rhonchus, indicative of bronchial irritation; inspiration rough and dry, intensity increased; the intensity and duration of expiration increased, and its quality blowing; slight bronchophony; diminished local fremitus; slight dulness, localized and distinct; heart's sounds transmitted; and in certain cases a subclavian murmur* (POLLOCK).

The following are the different modes in which phthisis pulmonalis makes its approach:—

1. There is sometimes to be noticed a latent, masked, or occult form of scrofula—the real condition of the patient not being detected till the lungs are tuberculous to a considerable extent, unless recourse be had to careful thermometric observation, as described in the first volume, page 32 to 51. Although the general symptoms may be slight, this very fact ought at once to excite suspicion, especially when the general aspect or constitution of the patient denotes the tuberculous cachexia. The slight cough, the shortness of breathing, the frequent pulse, increased on the slightest exertion, the languor of the frame, and general chilliness of the body with persistent increase of temperature, morning perspirations, and progressive

emaciation, betray the insidious way in which this form of consumption commences. Such a case must be closely watched, for the local symptoms often remain obscure, and friends do not always see the importance of the illness till too late. It is most usually young persons between sixteen, eighteen, and twenty-five years of age who are the victims. They begin to lose flesh, and are attacked with a short tickling cough, often regularly excited by undressing at bedtime, and again on getting out of bed in the morning. Such a cough is dry, or followed by only a small quantity of mucus. The cheeks are the seat of hot, uneasy flushings, while the feet are cold, and towards morning there is generally a little more moisture than usual on the surface of the body, especially about the head, neck, and breast. The pulse is either considerably quicker than usual, varying from 90 to 110 or 120; or if it is natural, it is very readily accelerated. The respiration is in general more frequent in a given time—usually from 24 to 28 in a minute—the inspiration being generally short, limited, and speedily checked, quickly succeeded by expiration; and the patient cannot take a full or deep inspiration without uneasiness, and without inducing coughing (CRAIGIE).

2. There is another form of consumption, attended with a severe and sudden accession of febrile disturbance (to be measured by the thermometer), occurring in persons of a scrofulous diathesis. To this form Sir James Clark has given the name of "*febrile consumption.*" Pulmonary symptoms are not generally manifest in such cases in the first instance; but what is commonly called biliousness more frequently prevails, or the case may present the symptoms of a common catarrh. Cough, however, generally soon appears, becomes urgent, and occurs often; and hurried breathing is one of the most remarkable symptoms. The cough speedily becomes more frequent, accompanied by expectoration, at first colourless, or of a thin, transparent, bluish jelly-like appearance; it subsequently assumes a yellowish or greenish hue, thick, opaque, dense, and puriform, and is occasionally streaked with blood. Fever continues unabated, and is out of all proportion to the other symptoms of pulmonary affection; and thus the true character of the disease may be overlooked. The pulse is seldom under 100, varying from 110 to 116, with some tension and sharpness in the beat. Gradually the fever assumes the hectic form, progressive wasting is established, and doubt can no

longer be entertained regarding the nature of the case and the fatal issue. (See range of temperature in "Acute Tuberculosis.")

3. In another description of cases hæmoptysis is the first symptom that attracts attention and alarms the patient and his friends. It is followed by all those phenomena already noticed.

The cases of phthisis more usually rapid are such as arise from hereditary constitutional causes, or from the influence of exanthemata, especially measles, or of typhoid or other fevers. These run their course with implication of several organs at an early stage of the disease. In the more chronic forms of phthisis the lung in adults is the first seat of the disease, and other organs are secondarily affected. In appreciating the causes of phthisis these forms must be distinguished (Parkes's *Practical Hygiene*, p. 444).

In the temperate zone, where the civilized inhabitants of the globe are located, it is calculated that *one-tenth* of the population die of this malady. Thus, in man, it is observed that in proportion as his habits of life are artificial, so is his tendency to scrofulous disease, especially of the lungs. This is strongly seen in the mining districts of Cornwall and Devonshire; for although those counties are considered among the most healthy portions of Great Britain, yet one-half of the whole number of the miners deprived of fresh air and light die of phthisis. In connection with this statement a most significant fact, of great practical importance, has been brought to light by Dr. Walshe. He has shown that improvement in cases of pulmonary phthisis was effected by medical treatment in hospitals, in about 14 per 100, more frequently in persons following "open air" and "medium" occupations than in those whose trades were "confined;" but that *death* or *deterioration* was as frequent as improvement in those who followed confined trades. Minute analysis, such as that which has been instituted by Dr. Walshe, of the numbers that die of phthisis in the different ranks and classes of life, is greatly to be desired in illustration of the remote causes of phthisis. The late Professor Coleman was of opinion that by confining the horse in a dark and dirty stable, and by feeding him on bad provender, and neglecting to clean him, he could produce phthisis on that animal at will; and similar causes will probably be found to produce similar results in man. When, however, we consider how many

persons there are who carry cleanliness to excess, whose diet is most studied, and whose every exercise is directed to health, and who nevertheless die of phthisis, it is plain that more secret and hidden circumstances still remain to be discovered to account for the existence of scrofulous disease, especially of the lungs, in this country. The Reports of the Registrar-General show that, comparing the deaths from phthisis among the agriculturists and among the inhabitants of towns, the latter die in an increased ratio of 25 *per cent.* over the former; yet it is generally supposed that the dietary and general comforts of the townsman are greater than those of the countryman. The chances of improvement are 5 *per cent.* greater in persons who come from the country to a salubriously situated hospital in town than in townspeople (WALSHE). Among townsmen, also, it is determined that there are certain classes of men more predisposed to phthisis than others by the nature of their occupations. It has been observed to occur for instance in those workmen who suffer great vicissitudes of temperature, or who breathe an air loaded with particles of dust: as bakers, needle grinders, stone-masons, quarrymen, cotton and wool carders, and bricklayers' labourers, and in this class of persons the disease has acquired the epithet of the "grinders' rot." As far as regards their state, much has been founded upon it to show how irritant substances may induce the local growth. The mortality numerically from consumption is much higher than from any other disease in this country; and amongst the class of society resorting to assurance offices the mortality seems to be about one-half of the whole.

The disease appears to be still more fatal to soldiers than civilians, producing nearly *one-half* of the whole mortality among the dragoon guards; while in the foot guards it has hitherto been nearly double of what takes place in the dragoon guards in this country. The habits of a soldier's life, his liability to febrile attacks, and the frequency with which he contracts venereal diseases, have hitherto been very favourable to the induction of the more aggravated forms of phthisis. The history of phthisis in armies will at once show how materially the prevalence of such a disease influences the health, the wealth, and the military strength of a nation. In Prussia, phthisis caused 27 *per cent.* of the total mortality; in Austria, 25 *per cent.;* in France, 22·9; in Hanover, 39·4; in Belgium, 30; in Portugal, 22 *per cent.* In all

these armies the same causes are in action, and the predominance of the disease is mainly to be sought for in the impure barrack air (Parkes's *Practical Hygiene*, p. 492). Some are inclined to ascribe this excessive amount of pulmonary disease and mortality to the night duties of the soldier—a statement in some measure supported by the large amount of mortality from consumption amongst night-watchmen generally; but when one looks to the age and height of the men enlisted for the regiments of the guards, and compares them with the physiological records regarding the stature and growth of the human frame, it will be seen how, sometimes, the combination of requirements for enlistment in the regiments of guards have been little calculated to secure a hardy and efficient body of men.* During 1859, 1860, and 1861, it appears that the mortality in the army hospitals from phthisis was not above that of the country generally, although it is clearly above that of the healthy districts; and there can be no doubt that there still is an excessive prevalence of tubercular disease in the army; and the astonishing disproportionate number of cases in the foot guards (18 to 20·6 per 1,000 of strength) is still as remarkable as it was twenty years ago (PARKES, l. c., p. 495). The tables prepared by Dr. Parkes, and given in his most valuable work on *Practical Hygiene*, clearly show that there must be a large amount of phthisis *generated* in the army; and in the foot guards nearly *four* times as much as among the civil male population of twenty-five to forty-five years of age.

The science of Medicine is not unfrequently indebted to non-professional people for correcting prevailing errors of belief and establishing correct opinions. No one, perhaps, contributed more in this direction, in the discharge of his own professional duties, than the late Sir Alexander Tulloch. It was long a prevalent belief that consumption was limited by latitude, and that it never appeared in warm countries—for instance, south of the Mediterranean. But this is proved not to be the case; for the returns of the army, prepared by the above writer, have shown that phthisis is more frequent in the West Indies than even in this country—a statement first made by Sir James Clark, in his work *On Climate*, in illustration of the injurious effects of that climate on consumptive patients sent there from this country.

* On this subject the reader is referred to a little book, by the Author, *On the Growth of the Recruit and Young Soldier*. Charles Griffin & Co., London.

According also to the recorded opinion of this author, great heat appears to have a powerful effect in predisposing to tuberculous diseases (probably by diminishing the exercise in the open air). That it is not the climate of the place which alone produces this result in the West Indies is shown by the fact that officers were attacked in infinitely smaller proportions than private soldiers; and in consonance with the views entertained regarding the nature of tuberculosis, it is more than probable that crowded barrack-rooms, a restriction to salt diet, and drinking spirits, may have produced the result.

It would appear that England and Wales, the Cape of Good Hope, and the Ionian Islands, are more exempt from phthisis than many countries which, from their higher temperature, have hitherto been supposed to enjoy a remarkable exemption from this complaint. The result of extended observation now entirely refutes the hypothesis that paludal districts are in an eminent degree exempted from phthisis—an opinion first promulgated by the late Dr. Wells, and advocated by M. Boudin. England and Wales, the Cape of Good Hope, Canada, and Malta—countries either the driest or the best drained, and consequently suffering the least from paludal diseases—are actually those countries the most free from phthisis. On the other hand, the influence of climate shows that phthisis is most frequent in low and damp situations; while it is far less so in the mountainous districts of all countries. Again, in whatever climate the disease breaks out, it is the opinion of many pathologists that its course is most rapid if the patient remains in that country; and therefore it is of the utmost importance to know the physical nature of the various climates of the world most suitable for the tuberculous patient (See Appendix to the third edition of Dr. Walshe's work *On Diseases of the Lungs;* and Sir James Clark's classic work *On Climate*). The late Dr. Hennen's experience convinced him, when the disease broke out among our troops on the shores of the Mediterranean, that no other chance remained of prolonging the patient's life than by at once sending him back to this country.

That the tuberculous constitution is transmitted from parent to child has long been a popular belief, and regarded as one of the best established points in the etiology of the disease. Actual *proof,* however, writes Dr. Walshe, has never yet been afforded of the justness of the general conviction; and as a step towards

an *accurate* settlement of the question, Dr. Walshe has analyzed and recorded (1849) the family history of 102 phthisical patients admitted into the Brompton Hospital for consumption. From these records it appears that about 26 *per cent.* of phthisical subjects in a given generation come of a tuberculous parent—a circumstance which may be predicated of any mass of individuals taken in hospital—namely, that 26 *per cent.* of them are of phthisical parents. On the other hand, while the general statement may be made, that some cases of phthisis may be traced to hereditary influence, it is undoubted that *much phthisis is, in each generation, non-hereditary.*

It further appears that in *males* the malady exhibits itself at a mean period of about two years earlier, and in females at a mean period of about three years and a half earlier when there is a parental taint than when there is not. Phthisical persons spring from a phthisical source with a certain amount of frequency; and that freedom from taint in parentage is *probably* more rare, and the existence of such taint *probably* more common, in phthisical than in non-phthisical patients; but it is possible that, if investigation was extended to infancy, childhood, and youth, the ratio of cases of parental taint among the phthisical would be proportionably greater than it proves where inquiry is limited to adults.

It appears, also, that while about 9 *per cent.* more phthisical than non-phthisical persons come of a consumptive father or mother, on the other hand, there are about 10 *per cent.* more phthisical than non-phthisical persons free from parental taint.

The final conclusion which Dr. Walshe arrives at, after a most careful and logical analysis of 446 cases of phthisical and non-phthisical cases, is this,—That *phthisis in the adult hospital population of this country is, to a slight amount only, a disease demonstrably derived from parents;* and there is no reason to believe that the law differs among the middle and higher classes of society. But amongst the phthisical cases which form the subject of Dr. Walshe's inquiry, we have the tuberculous cachexia communicated both as regards the parents and the generation following; but there is still a class of cases to be inquired into in a similar manner—namely, such as will show how far parents labouring under *scrofulous cachexia* merely entail on their offspring *a disposition* to tuberculous affections. It is now a well-known fact, emphatically insisted upon by Sir James Clark, that in the

families of consumptive parents there are constantly to be met with instances of ill-health characteristic of the scrofulous constitution; and in general such instances are much more frequent and much more strongly marked in the younger than in the elder children; nay, there are families in which the elder children are healthy, and the younger ultimately become the subjects of tuberculous disease. In such cases it has been presumed that in some instances the health of the parents has become deteriorated during the increase of their family. The mere fact of the parents being unhealthy, and not necessarily tuberculous, appears in some instances as if sufficient to entail tuberculous diseases upon their children. This statement is in some measure borne out by a result obtained by Dr. Walshe, that as far as the mere phthisical or non-phthisical condition of parents is concerned, about 24 *per cent.* of tuberculous patients can trace the origin of their disease to either parental source.

Contagion of Phthisis.—Regarding the possible contagious propagation of pulmonary phthisis, Dr. Parkes thus expresses himself (having regard to the fact that purulent and epithelial cells have now been demonstrated as floating about in the air where numbers of persons are together):—"Considering that the pleuro-pneumonia of cattle is probably propagated through the pus and epithelium cells of the sputa passing into the air-cells of other cattle; that even in man there is some evidence of a pneumonic phthisical disease being contagious (Bryson, *Cases in Mediterranean Fleet*), the floating of these cells in the air is worthy of all attention. It may explain some of those curious instances of phthisis being apparently communicated" (*Prac. Hygiene*, p. 74).

ACUTE PULMONARY CONSUMPTION.

Definition.—*Tuberculous growths or exudation in the lungs, expressed by febrile symptoms running an extremely rapid course, denoting the severity of the constitutional disturbance, proving fatal in from twenty days to ten or twelve weeks; and due to* (a.) *acute miliary tuberculosis, or* (b.) *acute pneumonic phthisis, or to both combined.*

Pathology.—Acute tuberculosis has usually been considered to be a comparatively rare disease; but it would appear that this fatal affection is unusually prevalent among the soldiers in Paris,

among whom also the chronic form of phthisis is very common, probably more so than even in our own army. In 1861, M. Colin (Professeur agrégé au Val-de-Grâce) records *five* cases of *acute tuberculosis* in the soldiers of the garrison of Paris (Parkes, *Army Med. Dep. San. Report*, 1860, p. 357).

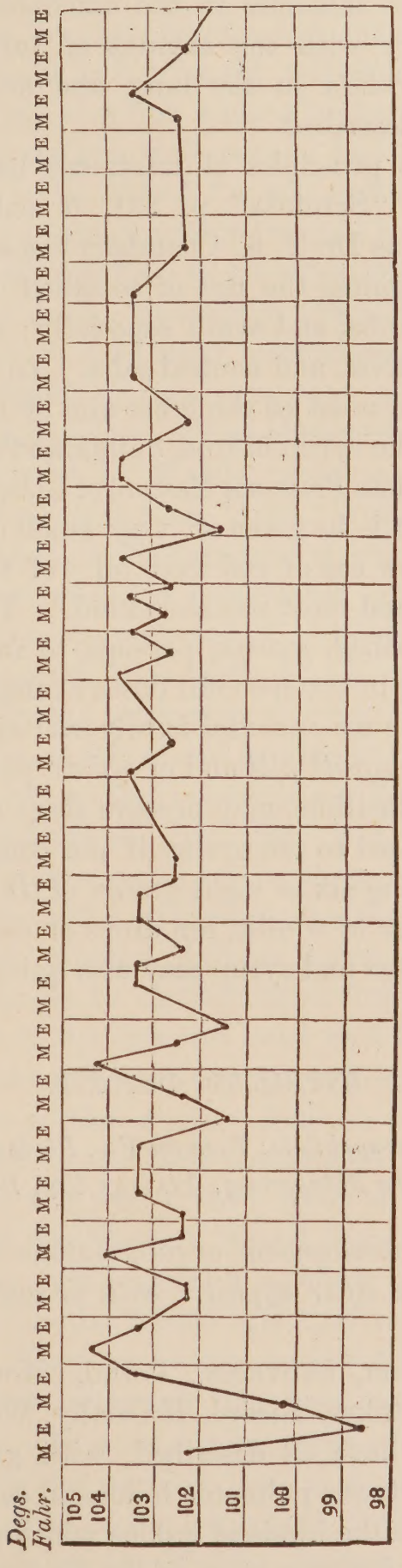
The disease occurs in two forms:—(1.) One form is connected with extensive growth or infiltration of yellow or crude tubercles of the lungs, with irregular softenings in the centres of some, or with small excavations surrounded by patches of hepatization. (2.) The form most characteristic of, or most commonly found in, acute phthisis is that in which there is general studding of both lungs with semi-transparent grey granulations, combined with pneumonia in its first stage, bright arterial injection, or hepatization (WALSHE). The two forms may co-exist in the same lung.

The Symptoms are those of an intense febrile affection. Dr. Parkes has favoured me with the notes of a case, exhibiting the correlation of the temperature, the pulse, and the respiration during the course of a fatal case of acute phthisis. The records of temperature are as in the diagram on next page.

During *thirty* days the pulse ranged from 116, the lowest, to 178, the highest, and having a mean of 140, reckoned by the number of observations. The respirations *per minute* ranged from 36 to 60, having a mean of very nearly 50. In Dr. Walshe's cases the relationship of the pulse to the respiration has varied considerably. The average has been as 3 to 1.

The functions of the lungs are deeply impaired. The invasion appears to occur while the patient is in a state of health, or the fever may be remotely preceded by various depressing influences, and immediate exposure to cold and wet; after which rigors ensue, followed by acrid heat of skin. The rigors recur on several successive days, followed by perspirations, and sometimes crops of sudamina. Epistaxis, followed by coryza, may occur on the second day of seizure (WALSHE). Prostration sets in early, so that in a few days the patient may be unable to stand. There is thirst, total anorexia, epigastric tenderness, dry lips and tongue, dental sordes; all signifying great intestinal disturbance. Diarrhœa is rare, and constipation may be extreme, even with abdominal pain and ulcerated intestines. Restlessness, *insomnia, cephalalgia, vertigo, tinnitus aurium,* diurnal wandering and nocturnal delirium bespeak cerebral complications and the

DIAGRAM SHOWING THE RECORDS OF TEMPERATURE IN A CASE OF ACUTE PHTHISIS (Parkes).


Degs. Fahr.
105
104
103
102
101
100
99
98
M E M E M E

probable growth of tubercles in the arachnoid membrane. The physical signs vary with the amount of tubercular growth or *pneumonic infiltration* in the lung, and are similar to those which have been described.

Treatment.—The principles of treatment having been already laid down under "Scrofula," p. 221, it only remains here to warn the student (as Dr. T. K. Chambers has so ably done in his Lectures, p. 270) against the use of so-called "cough medicines," antimony, ipecacuanha, and squill especially; also against the use of mercury, purgatives, and neutral salts. To foster and cherish an appetite for food must be the great aim of treatment. One of the best tonics is the syrup of iron, quinia, and strychnia, of which the formula by the late Professor Easton, of Glasgow, has been given at p. 62, *ante*. Let it be given in very small doses at first; and followed up by the use of cod-liver oil "of the most agreeable, clearest, sweetest, and most scentless kind." The *brown oil* ought never to be prescribed, except, perhaps, to an Esquimaux. The oil should be given in tea-spoonful doses at first. The old *vinum ferri* in doses of one tea-spoonful hardly ever disagrees: and equal parts of *compound squill pill* and of *opium pill* (P. B.), given in *five* grain doses at bedtime, may procure sleep at night; and the dose may be increased to ten grains if the cough is troublesome. A mixture containing six or eight grains of *Dover's* powder, with ten drops of *tincture* of squills, ten drops of *tincture* of *Tolutan*, with a little mucilage and syrup may also quiet the cough.

SYPHILITIC DEPOSIT.

LATIN EQ., *Deposita ex syphilide;* FRENCH EQ., *Dépôt syphilitique;* GERMAN EQ., *Syphilitische Ablagerung;* ITALIAN EQ., *Deposito sifilitico.*

Definition.—*The development of gummatous nodules in the substance of the lungs after syphilis, with or without bronchitis or miliary tubercle.*

Pathology.—Morton, Sauvages, Portal, Morgagni, and more recently Graves, Stokes, Ricord, M'Carthy, Walshe, Wilkes, Virchow, and Munk, have all described, with greater or less uniformity and distinctness, pulmonic lesions in cases of syphilis.

The following are the kinds of lesions which predominate:—

1. *Evidences of Inflammation of the Mucous Membrane of the*

Bronchial Tubes.—In such cases bronchial irritation, with fever in many cases, precedes the skin lesions, and may disappear wholly or partially when the skin lesions are established. On the other hand, if the syphilitic eruption suddenly disappears, bronchitis may ensue. Walshe records well-marked instances of this; and it is a circumstance to be looked for amongst soldiers especially, who, having recovered from a primary syphilitic sore, are apt to be exposed to the risk of bronchitis when mounting guard soon after being discharged to duty. Secondary symptoms and pulmonic lesions are then apt to date their commencement; and all the general symptoms of phthisis may supervene, and yet no tubercle in the lung may be developed; but chronic bronchitis remains persistent. On this point Dr. Walshe observes, that in the persistence of the general symptoms there "is assuredly enough to create a strong suspicion of the existence of tubercle in the lungs, taken in conjunction with the indubitable tendency of syphilis *plus* mercury to induce the outbreak of phthisis in a person having the requisite constitutional aptitude. How are the cases to be distinguished? By the total want of accordance between the physical signs and the constitutional symptoms: the patient with syphilitic bronchitis has neither consolidation signs nor, *à fortiori*, the evidences of excavation. But there is a curious source of difficulty which sometimes starts up in these cases, and renders doubt imperative—the infra-clavicular ribs and clavicle thicken from periostitis, and produce dulness under percussion, which cannot with positiveness be distinguished from that of tubercle within the lung. Here the observer must wait for events to clear up the diagnosis" (*Diseases of the Lungs*, p. 233).

2. *The Occurrence of Gummatous Nodules in the Pulmonary Substance.*—These are, in the first instance, of the same histological constitution as the well-known node of the shin, or the subcutaneous product described by Ricord, Bärensprung, Virchow, and M'Carthy. They form especially towards the periphery and bases of the lungs. In the former site they resemble nodules of lobular pneumonia. They may soften and be eliminated much in the manner of tubercle, although they may have at first a consistence like scirrhus. It is concerning those which soften in this way that Ricord gives the warning "not to confound suppuration of a few syphilitic nodules of the lungs with phthisis." Regarding those gummatous nodules Dr. Walshe observes: "I can find no

positive answer to the query, Do these gummata ever form independently of other tertiary evidences of syphilis in the bones and cellular tissue? If they do, their diagnosis must be infinitely difficult—difficult, indeed, under all circumstances; for the physical signs can be none other than those of solidification, followed by softening and excavation, while the local and general symptoms closely simulate those of phthisis" (*Diseases of the Lungs*, p. 431).

3. *The Occurrence of Gummatous Nodules in various Stages of Growth and Degeneration, associated with the Miliary Deposit of Tubercle.*—In such cases the history of events in the illnesses of the patient may be found to correspond more or less closely with the appearances seen in the lungs—appearances which distinctly indicate the formation of lesions commenced at different dates—appearances which denote the occurrence of lesions in crops, or as a succession of events which may be illustrated by the history of the symptoms during life.

The minute structure of these *gummatous nodules* has been closely examined by many observers. They consist of a growth of elements which leads to the development of an elastic tumor composed of a well-defined tissue, and the elements of which are extremely minute. The tumor takes origin from the connective tissue, or the analogues of such; and hence the universality of the site of syphilitic lesions. When these are sufficiently large to attract attention—as in the form of a node on the shin bone, or on some part of the true skin—they are small, solid, pale knots, like a hard kernel, about the size of a pea. They are generally first seen on some part of the true skin or subcutaneous or submucous tissue; and when the tissue in which they happen to grow is sufficiently lax, they grow to a considerable size, and convey to the touch a sensation as if they were filled with gum. Repeated examinations of this growth show that in its gelatinous or soft state it arises from a proliferation of nuclei amongst the elements of the connective tissue, not unlike the formation of granulations in a wound. The component cell-elements appear as round, oval, or oat-shaped particles imbedded in a matrix of fine connective tissue of a granular character, and tending to fibrillation. The cell-elements are a little larger than blood-globules, and are distinctly granular in their interior when mature. In the growing part of the node, and immediately in its vicinity, where growth is abnormally active, the minute cell-elements are seen to

be developed in groups within the elongated and enlarged corpuscles of the connective tissue. In form, therefore, the node or gummatous nodule resembles a tubercle; and by fatty degeneration or tuberculization may not be capable eventually of being distinguished from tubercular deposit. How, then, are we to recognize the specific nature of such gummatous nodules? There is nothing in them so specifically and anatomically distinct that, apart from their history, they can be recognized. The history of the syphilitic case during life is the great guide. The nodes on the shin-bone or clavicles have long been recognized as the product of syphilis. It may almost be said that they have been seen to grow under the eyes of the patient and the observer; and their anatomical characters are found to be such as compose the gummatous nodules just described. In a case of inveterate syphilis, therefore, whose history is fully known, in whom the node on the shin or other bones is characteristic, and has been seen to grow, and in whom also we find similar nodules in the lungs, or in the liver, or in the testicles—symmetrically growing in these latter organs, and consisting of minute cell-elements exactly the same as the node on the shin—it is impossible to overlook the fact, or not to be impressed with the belief, that all of these lesions acknowledge one and the same cause of development—namely, the syphilitic poison—of which they are the expression. The progress of the node is also characteristic and suggestive. Growths of a similar form which result from idiopathic inflammation generally proceed to the formation of an abscess, or to the hypertrophy of fibrous tissue. Abscesses are recognized by their pus; fibrous tumors or hypertrophies, by the fibre-elements which compose them.

Growths of a form similar to the node, which result from cancer, are in general to be recognized by the juice expressed from them. In the gummatous nodule we have no juice, and the cell-elements seen in cancer are generally so diversified in their form and mode of growth as not to be easily mistaken. The gummatous nodule is uniform as to the size and form of its cell-elements, and forms a growth less highly supplied with blood-vessels than a cancer. Cancers tend to infiltrate and involve neighbouring textures; the gummatous nodule remains isolated and distinct.

By way of elimination, therefore, and by duly observing the

history of the case, we are generally able to recognize the nature of such growths, and to assign to them their proper place in pathology.

The gummatous nodule has now been recognized and described in almost all the solid viscera of the body. Symmetrical development is a most constant characteristic. If a node grows on one shin, it is probably also to be found advancing on the other; if found in one testicle, it is extremely probable that it will be seen in the same relative spot in the other. Numerous examples of this symmetrical development may be seen preserved in the Pathological Museum of the Army Medical Department at Netley. During the growth of the nodule, proliferation advances slowly, and a gluey-like material forms, which constitutes the inner cell-material of the nodule. If near the surface, such a nodule is apt to melt down, soften, open, and ulcerate; and such a result seems to be associated with other evidence of active constitutional disease, such as exists with a predisposition to tubercle, or with its actual existence. The tumor, however, continues gelatinous and coherent if it is enclosed in a dense part, or is deeply seated, as in gummata of the periosteum, scalp, brain, liver, testicle, lungs, and heart, if constitutional disease remains latent or inactive. Fatty degeneration may also eventually occur in the gummatous nodule, and eventually lead to its absorption; or its absorption takes place as a natural process of cure, the changes of which are not exactly known. We know only that the node on the shin-bone not seldom disappears from view, and does not return.

For reasons already stated, phthisis must be regarded in many cases as the product of syphilis; and I would fully endorse the statement of Dr. Balfour, from what I have seen in the *post-mortem* rooms, when he says that a great cause of pulmonary disease among the Guards is the amount of syphilis which prevails amongst the men, which he has not the least doubt is a very fertile cause of its being called into active operation. The influence of syphilis on the health of the soldier is indeed powerful for evil throughout the whole army.

Treatment.—The treatment of such pulmonary lesions, when their nature is clearly established, must be guided by the rules already laid down for the treatment of syphilitic disease at p. 780, vol. i.

GANGRENE OF THE LUNG.

LATIN EQ., *Gangræna;* FRENCH EQ., *Gangrène;* GERMAN EQ., *Brand*—SYN., *Gangrän;* ITALIAN EQ., *Gangrena.*

Definition.—*Disintegration and breaking up into fragments of the filamentous tissue of the lung. Every simple element of the tissue is so changed that neither blood-fibres nor epithelium can be recognized in a sound state. The texture becomes broken up, and shreds of fibrous tissue may here and there be distinguished, but the whole mass becomes converted into a heap of amorphous granular matter, of a yellowish-brown or black colour, mingled with drops of oil. The tissue becomes soft and flaccid, in some parts perfectly liquescent, and generally emits a fetid smell.*

Pathology.—The comparative rarity, the almost invariable fatality, of pulmonary gangrene, obtains for it a melancholy interest whenever it occurs; and since Laennec directed attention to its peculiar characters, many pathologists have contributed to the records of this disease. But notwithstanding the researches of Andral (1822), of Lorimer, of Schroeder Van der Kolk (1826), of Bright (1827), of Cruveilhier (1833), of Guislain (1836), of Craigie (1841), of Ernest Boudet (1843), of Silfverberg (1856), of Walshe (1860), of Fuller (1862), gangrene of the lung is still a condition at all times difficult of diagnosis in the first instance; and its existence has often been unknown until the disgusting *post-mortem* appearances proclaimed the condition, which general symptoms during life had failed to disclose, and which in some cases hardly even suggested a suspicion of the real nature of the affection.

The diagnostic symptoms which have been most frequently recorded are derived from the expression of the countenance, becoming small, pinched, contracted, haggard, ghastly, miserable, and death-like; eyes sunk and void of lustre; patient squeamish and languid, with occasional vomiting, and a feeling of indifference to all external objects,—some or all of which symptoms may or may not be associated with an intolerable fetor of the breath, and which, when it is present with these symptoms, may be considered conclusive of the existence of gangrene. Not one of all these symptoms, however, may manifest themselves, and yet gangrene of the lung may exist. From the recorded cases,

therefore, as well as from those which I have myself seen, it may be of some use to classify the conditions of disease in which pulmonary gangrene has occurred.

The termination of acute sthenic pneumonia, in an otherwise healthy person, by gangrene of the lung, is by all authors considered as one of the least frequent terminations of that disease. Morgagni only records one instance; and Laennec is reported to have seen only six or eight during the whole course of his practice. But there can be no doubt that pneumonia does sometimes terminate in gangrene of the lung, as an accidental or occasional complication, as has been clearly shown by clinical and *post-mortem* evidence. The insidious and often sudden mode of its attack, the sudden, remarkable, and generally fatal collapse which supervenes, show, at the same time, that no local condition is suspected or considered sufficient to account for the presence of gangrene. The extent to which the death and destruction of lung-tissue takes place also varies much. Sometimes nearly the whole of a lung passes at once into a gangrenous mass. In other cases only a small portion in the centre of an exudation will die; but in every case there is the formation of a slough, its liquefaction, and the formation of a cavity. Sometimes a line of separation between dead and living parts is attempted to be formed; while in other cases no evidence exists of any limit to the extent of the destruction; and this latter diffuse form of gangrene I have generally found associated with cases in which the blood was greatly changed, as in typhus fever, or in which the nervous functions were impaired, as in maniacs, or in the paralysis of the insane. Of fifteen cases recorded by Guislain, all were maniacs. Of three cases recorded by Van der Kolk, one was a maniac and two laboured under some peculiar nervous condition of the body. Of six cases recorded by Dr. Craigie, two were mentally deranged, having lesions in the brain; two suffered from *Bright's disease* and *mercurialism;* one had variola; and one had typhus. Of fifteen cases in the Royal Infirmary of Edinburgh when I was a student there, four had typhus; three had fever, the type of which is not specified; four suffered from ill-health and mercurialism; one suffered from a blow; and one had a lesion in the brain.

These cases may be classified as follows:—

Nineteen cases were associated with, and doubtless influenced by, lesions in the great nervous centres; and under this class are included the insane.

Nineteen cases were associated with, and doubtless influenced by, such morbid conditions of the blood as exist in *typhus fever, variola, tuberculosis, Bright's disease, mercurialism.*

One case was associated with and influenced by arterial obstruction, causing pressure upon the entire mass of the affected parts, and total absence of circulation in them (Aitken, *Edin. Med. and Surg. Journal,* No. 178, 1848).

The frequency with which lesions of the lung co-exist with and succeed to lesion of the brain renders it apparent that the condition of the lung is materially affected by the influence of the nervous centres. In the cases recorded by Andral and Bright, diseases of the lung, frequently going on to gangrene, were often the immediate cause of death in persons suffering from cerebral disease; and it was also observed that the tendency of inflammation of the lung to pass into gangrene was promoted by the existence of disease of the brain. Cruveilhier also directed attention to the frequency of gangrene of the lung in epileptic cases; and the insane generally are prone to the disease when the bodily health has suffered, or where, as in maniacs, a greatly depressed state of the animal functions succeeds to inordinate nervous excitement.

The following table, modified from that by Dr. Walshe, describes the conditions under which gangrene of the lung occurs:—

I. Pulmonary diseases.	*Pneumonia*, acute and chronic; tuberculization; cancer; hæmorrhage; hydatids (Walshe); apoplectic foci in the lung; acute and chronic dilatation of the bronchial tubes; pleuritis; bronchitis (Silfverberg).
II. Other thoracic diseases.	*Cardiac:* acute endocarditis of the right side of the heart. *Mediastinal:* tumor. *Aortic:* aneurism.
III. Diseases in which the blood is materially changed.	*Animal venoms:* stings of certain insects. *Morbid poisons:* Glanders; exanthemata; typhoid fever; Bright's disease; alcoholism. Scurvy; purpura; septicæmia; pyæmia. *Other poisonous agents:* poisonous gases; mercurialism.
IV. Chronic abdominal disease (Silfverberg).	
V. Perverted innervation.	Epilepsy; insanity; organic cerebral disease.
VI. Traumatic.	

The disease may occur at any age; but it appears to be more common in children than in adults; and more so in adults than in persons much advanced in life.

The symptoms vary according as the lesion is *diffused* or *circumscribed*:—(1.) In the diffused form the symptoms and progress of the case are extremely rapid. Utter prostration of strength; oppressed breathing; a small, weak, and frequent pulse; pallor and anxiety of countenance; and all the general appearances of intense adynamia, are those which characterize the disease. There is also frequent but feeble cough, with profuse, frothy, and diffluent expectoration, of a peculiar greenish colour and intensely fetid gangrenous odour. But the power to expectorate is soon lost; and ere long the vital powers seem utterly oppressed or exhausted; the pulse fails, the features collapse, and the patient rapidly sinks, death generally occurring from apnœa. (2.) In the *circumscribed* form of gangrene the train of symptoms varies greatly at different periods of the disease. At first, signs of pulmonary congestion are coupled with an amount of prostration quite out of proportion with the apparent extent of local lesion—a prostration which is, indeed, sometimes the only prominent feature in the case. After a time expectoration commences; at first muco-purulent, rarely bloody in adults, frequently so in infants and children (if they expectorate at all), and generally emitting a disagreeable odour the moment a communication is established between the bronchial tubes and the dead tissue. The sputa then lose their muco-purulent character, and become extremely liquid, sero-purulent, and of a dirty greenish, yellowish-brown, or ash-grey colour. They exhale an odour distinctly gangrenous, while the breath acquires the same offensive putrid smell, resembling somewhat that of wet mortar, or its fetor is one *sui generis*. The pulse becomes feeble and rapid, with every evidence of great and increasing prostration. The patient passes rapidly into a state of collapse, and sinks in a few hours or days, without the occurrence of any other change, unless profuse hæmorrhage occurs, which terminates in death (Walshe, Fuller).

Treatment.—The chief reliance is to be placed in all forms of stimulants, combined with such tonics as *bark* or *quinine* in full and repeated doses. Dr. Walshe recommends that repeated doses of an ounce of yeast are deserving of investigation, as well as the

influence of *chlorate of potass.* He is disposed to put more confidence in them than in ammonia, the beneficial effects of which he has never seen demonstrated. Dr. Fuller has not found ammonia so useful as the mineral acids. Inhalation of the vapour of turpentine poured upon boiling water, as recommended by Skoda, is reported to exercise a distinctly remedial power. The *mineral acids,* especially *nitro-muriatic,* combined with *quinine,* are the main remedies in the chronic state of this disease. A generous diet, easily digestible, with as much malt liquor as can be taken, are recommended as most suitable for such cases.

SPITTING OF BLOOD, PULMONARY HÆMORRHAGE—Syn., HÆMOPTYSIS.

Definition.—*The discharge of blood by expectoration.*

Pathology.—The amount expectorated may be small—mere specks or streaks on the sputa; at other times a few ounces, or even pounds, causing great alarm to the patient or his friends, and sometimes suffocating the patient. Its occurrence is always suggestive of organic disease of the chest, such as engorgement of the pulmonary vessels, giving way of pulmonary tissue, indicating disease of the heart, or pulmonary tuberculosis or pneumonia, or thoracic aneurism, or cancer of the lung. Cases are also recorded of its so-called idiopathic occurrence, as from variations (sudden) of atmospheric pressure, ascending high mountains, or descending in diving-bells, violent straining efforts, or from plethora; but in such cases, according to the experience of Drs. Fuller, Walshe, and others, "there is usually some latent mischief in the chest—some local cause of pulmonary congestion—some mechanical interference with the capillary circulation through the lungs." Spitting of blood may even precede for years the fatal development of tuberculous disease, with which it seems to be most prevalently connected, and is often hereditary.

Symptoms.—Hæmoptysis may take place suddenly, or be preceded by a sense of heat or a feeling of weight at the chest, or the patient may suffer pain between the back and shoulders, or may labour under dyspnœa, palpitation, cough, or coldness of the extremities; and these symptoms may last two or three days. At length a fit of coughing, or a tickling of the throat, is followed by the appearance of expectoration of arterial, but more often of venous blood. Laennec says he has seen as much as ten pints thrown up

in forty-eight hours, and as much as thirty pints in a fortnight. The effort of coughing also often causes vomiting, so that the blood discharged is frequently mixed with alimentary matters.

If the quantity thrown up be inconsiderable, the patient's previous health is in no degree affected; but if it be large, its effects are strongly marked, for the patient feels oppressed at the præcordia, breathes with difficulty, and with a gurgling sound, caused by the air passing through the viscid blood retained in the bronchi; and this is shortly followed by increasing weakness, even to complete prostration. In still severer cases, as the blood flows, the patient turns pale, his countenance becomes œdematous, and strongly expressive of terror, or else he falls into a complete syncope, which sometimes has a curative effect. In a very few instances the effusion is so sudden and so considerable that the patient dies suffocated.

Although bronchial hæmorrhage may be considerable, it often diminishes rapidly, so that at the end of some hours only a few rare isolated sputa are spat up, and at considerable intervals. Usually, however, the hæmoptysis recurs after a greater or less length of time, but not perhaps to the extent of the primary attack. After the patient has lain for a time in a state of depression, a re-action takes place. In sthenic persons the appetite becomes increased, they enjoy everything they are allowed to eat, and after some slight febrile action they rapidly recover. In the fatal cases the pulse becomes rapid, the tongue brown and dry, and the patient sinks.

Diagnosis.—The only disease which it is important to distinguish from *hæmoptysis* is *hæmatemesis*, and the diagnosis between them is difficult, because while the contents of the stomach are always rejected in *hæmatemesis*, they are frequently rejected in hæmoptysis also. The stethoscope, however, greatly assists in determining the seat of the disease; and again, blood is generally found in the stools in cases of *hæmatemesis*, while it is for the most part wanting in *hæmoptysis*.

Prognosis.—The prognosis is always unfavourable eventually, so far as freedom from organic disease is concerned, and danger is more or less directly imminent in proportion to the amount of blood lost.

Treatment.—The medicines most useful in *hæmoptysis* are the *bitartrate of potash, in doses of a drachm*, repeated every four or six hours, and to each dose of which may be added a quarter to half a grain of *opium*. The *mineral acids*, as the *infusion of*

roses with diluted *sulphuric acid*, in doses of from three to five drops, combined with *opium* or *morphia*, every four or six hours; larger doses of the *dilute sulphuric acid* have often been tried, but are apt to be either rejected or to act injuriously on the coats of the stomach. Many practitioners use one to three grain doses of the *acetate of lead* every four or six hours, with *half a grain of opium* to each dose, or combined with *dilute acetic acid* and *laudanum* (A. T. THOMPSON); and, according to Andral, when the system has long been under the influence of lead, the red globules suffer a great diminution; but, nevertheless, this is certainly a less efficacious medicine than either of the preceding ones. The *nitrate of potash* has been much used in France, but Gendrin has not found it efficient, or not more so than any other diuretic. The *muriate of soda* in doses of half a drachm to a drachm is in estimation with some practitioners on the Continent.

When hæmoptysis is connected with amenorrhœa, preparations of iron often succeed when the above remedies have failed. Two grains of the *sulphate of iron*, with one drachm doses of the *sulphate of magnesia*, three times a day, often restore the menstrual secretion and cure the hæmoptysis. Indeed, it is in this form of amenorrhœa that iron is most successful.

When hæmoptysis depends on disease of the heart, cupping from the chest, or moderate bleeding from the arm, is often efficacious, combined with the use of the *bitartrate of potash* or the mineral acids, to which should be added five to ten minims of the *tincture of digitalis*. It is in many cases proper to add half a drachm to a drachm of the *spirit of nitrous ether* to each dose, to give steadiness to the irregular, turbulent, or rolling action of the heart.

Dr. Fuller's experience leads him to testify most strongly in favour of repeated dry cupping, aided by the application of ice down the spine, and by the internal administration of *full* doses of *digitalis* (℥i. to ℥iii. of the tincture; or gr. vi. to gr. viii. of the powder).

If these remedies fail, full and frequent doses of *gallic acid*, or *lead and opium*, may be given, if the circulation is much accelerated, and of *spirits of turpentine* in half drachm doses, if the bleeding is unattended with vascular excitement. The *gallic acid* should be given every hour in *eight* or *ten grain* doses, until the hæmorrhage is subdued, or till a dark green colour in the sputa indicates its action in the system (*op. cit.*, p. 265).

Absolute bodily and mental rest must be insisted on during convalescence.

CHAPTER XI.

DISEASES OF THE MOUTH, TONGUE, FAUCES, PHARYNX, LARYNX, AND ŒSOPHAGUS.

SECTION I.—AIDS TO THE DIAGNOSIS OF DISEASES OF THE MOUTH, FAUCES, PHARYNX, AND LARYNX.

THE principle of the speculum in use by dentists, and first used by Robert Liston, the eminent British surgeon, more than twenty years ago, in the diagnosis of an "ulcerated glottis," has greatly advanced our knowledge of the lesion of parts otherwise beyond

Fig. 36.*

our vision. Science is indebted especially to Garcia, Czermack, and Turck, in Germany, and to Drs. Morell Mackenzie, Gibb, Sieveking, Johnson, and Walker, in this country, for promoting and making known the practical application of the laryngoscope.

* Laryngoscopic examination of a patient (after DR. GEORGE JOHNSON).—The illumination of the pharynx is effected by light cast into the patient's mouth from a mirror over the brow of the operator. Dr. Johnson has recently suggested that the operator should also use a shade to shield his eyes from the glare of light (*Lancet*, August 26, 1865).

The Laryngoscope consists of a small flat mirror with a long stem, which, being previously warmed, to prevent the breath condensing upon it, is introduced into the widely open mouth, as far back as its back part. In the application of the instrument the first great difficulty to overcome is the illumination of the pharynx; the second difficulty is to learn to manipulate the mirror in such a way as to display the larynx, or any part it is desired to see, to the best advantage (fig. 36). It must be remembered that the image of the parts are reflected to the eye of the observer in *an inverted position*, so that the right vocal cord appears on

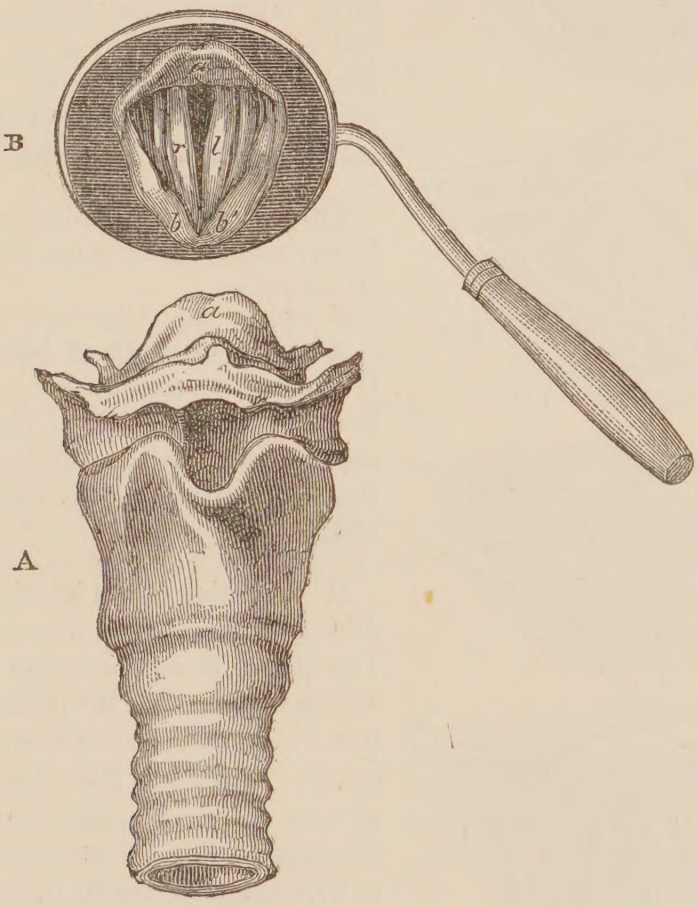


Fig. 37.*

the left side, just as the right arm appears on the left in a looking-glass. Experience will soon enable the observer to correct this falsity of representation, and he will do this quite unconscious of any mental effort (fig. 36). By means of this instrument the deep portion of the pharynx, the larynx, the passage through the glottis, the mucous membrane of the trachea, are all

* A. Anterior view of the larynx.—(*a.*) *Epiglottis.* B. Opening of larynx, as seen inverted in the mirror.—(*a.*) Epiglottis; (*b*, *b'.*) Arytenoid cartilages; (*r* and *l.*) Right and left vocal cords (after T. J. WALKER).

capable of being seen. The epiglottis and posterior aspect of the base of the tongue, as well as the posterior aspect of the pillars of the *fauces*, and the condition of the *nares*, are all capable of being seen in this way, as represented in the following wood-cuts, figs. 38, 39, 40, 41:*—

* A, B, C, D, E, represent the arrangement of the internal parts of the larynx of Dr. Czermack, after drawings made by Dr. Elfinger, of Vienna, from images demonstrated in the laryngoscope (*New Syden. Soc. Year-Book*, 1861). The parts situated *to the right* of the middle line of these figures, obtained by the aid of a mirror, are necessarily reversed from their natural position, and correspond to those *on the left* side of the larynx, and *vice versâ*. That which is situated above in the drawing exists in front; that which is below is situated posteriorly.

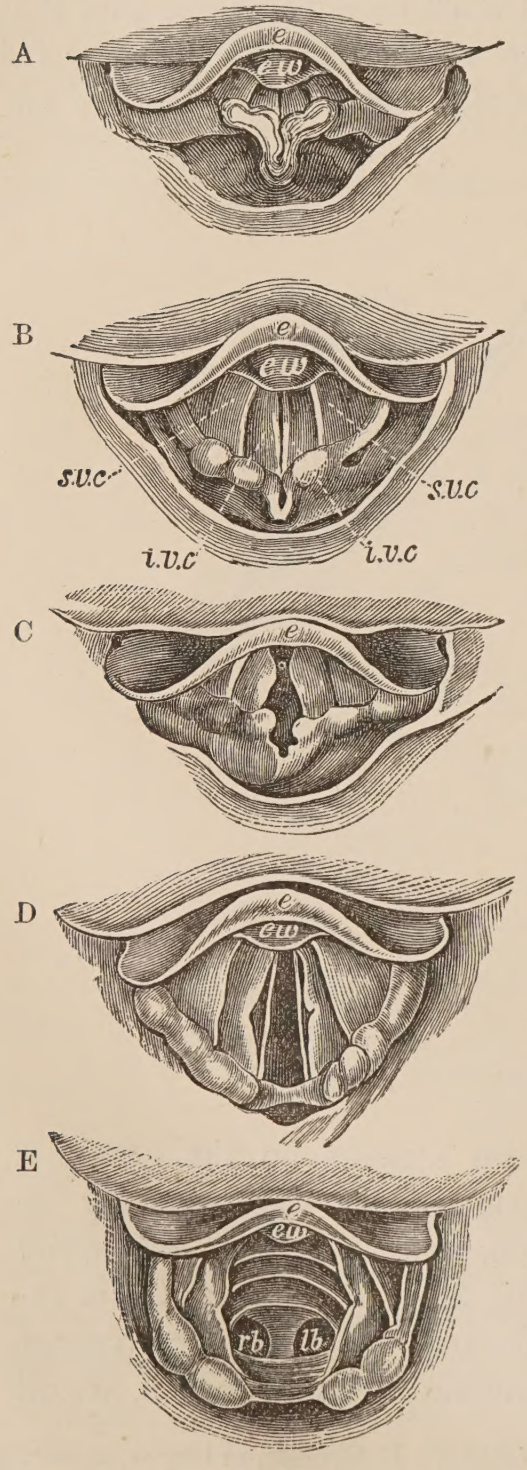


Fig. 38.

The series of Wood-cuts show the Larynx from Moderate Closure to full Dilatation.

A. The beginning of hermetic closure of the larynx—commencement of act of swallowing. The cushion of the glottis (*e*, *w.*) applies itself over a considerable portion of the closed glottis, and of the narrowed false glottis.

B. Condition of the interior of the larynx during the production of most acute sounds. The glottis is contracted in a linear form; and on each side are the (inferior) vocal cords (*i*, *v*, *c.*), distinguished by their whitish-yellow colour. A little to the outer side is a narrow groove, which indicates the ventricles of Morgagni. Farther outwards are the false or superior vocal cords, (*s*, *v*, *c.*)

C. Constriction of the glottis, erroneously called *respiratory glottis*, the arytenoid processes converging, and causing the posterior section of the glottis to be almost triangular.

D. Complete relaxation of all the parts, as when the glottis under ordinary circumstances is open for breathing.

E. Bifurcation of trachea and commencement of bronchial tubes, seen on widely opening the glottis, as in deep inspiration, and straightening the trachea.

The instrument is absolutely necessary, not only for precision of diagnosis, but for precision in the local appliances in the treatment of diseases of these parts. The application of *glycerine*, of *nitrate of silver*, and other agents, can thus be applied with perfect accuracy to a part. The reader is referred to the papers of Dr. Morell Mackenzie in the *Medical Times* for April, 1862; to those of Dr. Sieveking in the *British Medical Journal* for December 20, 1862; and to those of Dr. T. J. Walker in the same journal for

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APPEARANCES OF THE LARYNX AS ALTERED BY DISEASE (after CZERMACK):—

F. Condylomata of the larynx, believed to be the result of syphilis. Conical excrescences on inferior vocal cord, with thickening, causing hoarseness.

G. Polypus attached to the right vocal cord, the real cause of a supposed venous aphonia.

H. Transformation of the right (superior) false vocal cord into a hard, rough, and ulcerated mass.

K. Cicatricial contraction and loss of substance of the larynx, with destruction of epiglottis from old syphilitic disease.

L. Very large muciform polypus, of an epithelial character, developed within and above the glottis—aphonia of four months.

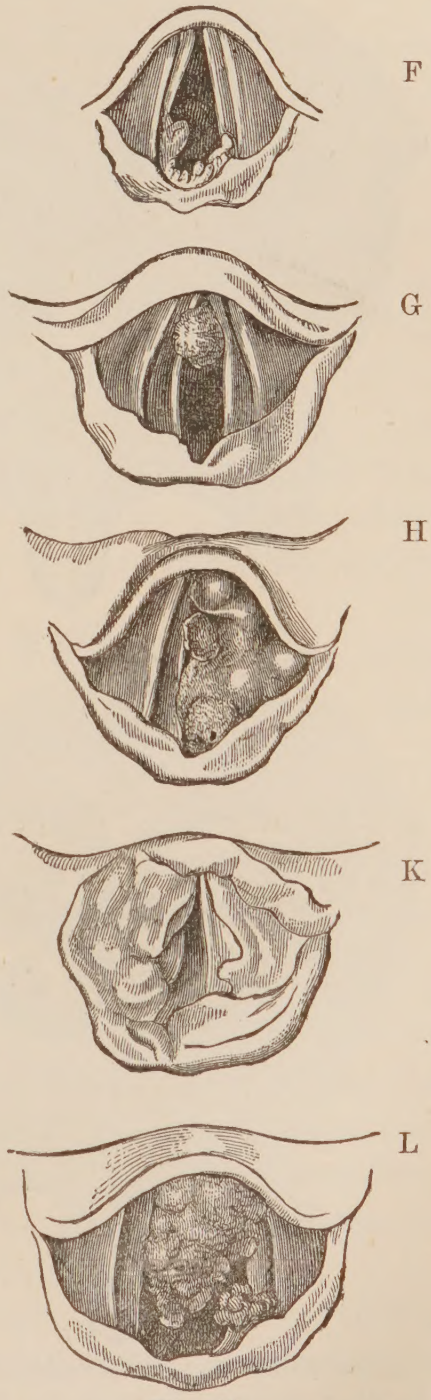

Fig. 39.

February 28, 1863, *et seq.*, and to the separate monographs by these writers, for an account of the methods of using the instrument.

SECTION II.—DETAILED DESCRIPTION OF THE DISEASES OF THE MOUTH, TONGUE, FAUCES, PHARYNX, LARYNX, AND ŒSOPHAGUS.

INFLAMMATION OF THE MOUTH—SYN., STOMATITIS.

The mouth is specially liable to various forms of inflammation

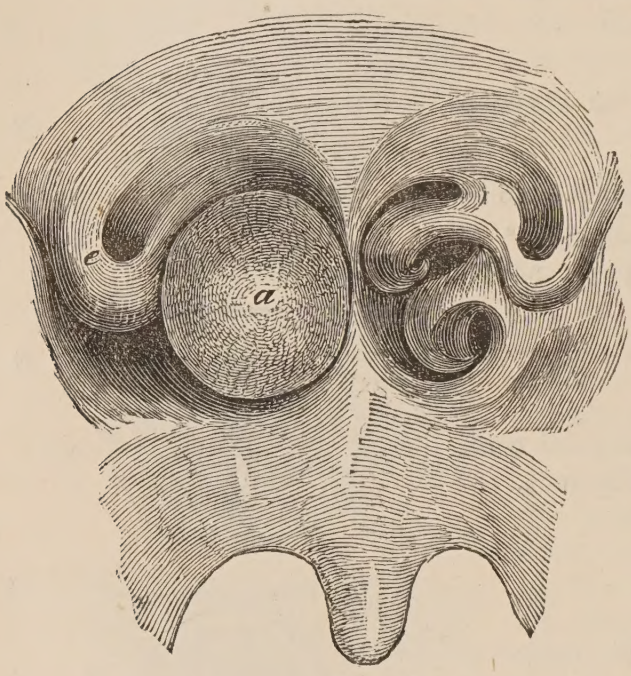

Fig. 40.

INSPECTION OF THE NARES.—*Rhinoscopy.*

Posterior view of uvula, soft palate, and nares, as seen in the mirror (directing it upwards in place of downwards). The tumor (*a.*) on the *left* side of the figure fills up the posterior opening of the *right* nasal forsa; (*e.*) The orifice of the right Eustachian tube (after DR. GEORGE JOHNSON).

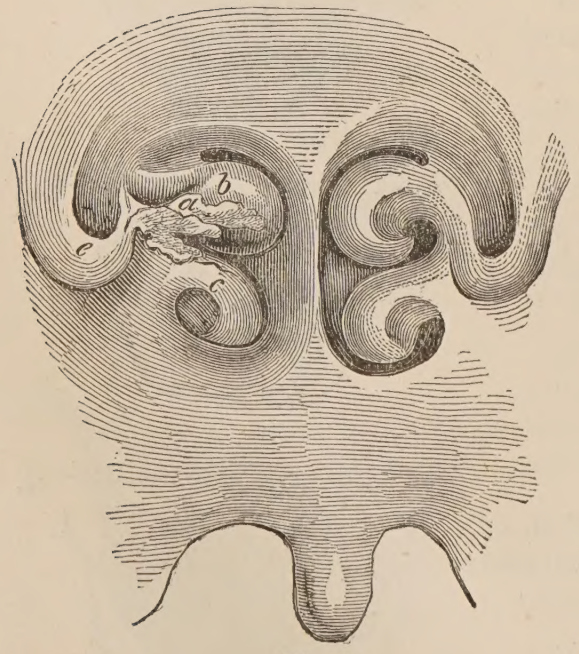

Fig. 41.

Posterior view of the parts after removal of the tumor. (*a.*) The abraded surface from which the tumor was torn; (*b.*) The middle; and (*c.*) The inferior turbinated bones; (*e.*) The Eustachian tube (after DR. GEORGE JOHNSON).

[These drawings, illustrative of the larynx and laryngoscopy, were kindly made by Staff Assistant-Surgeon Dr. Humphrey C. Gillespie.]

in children. They have received the name of "stomatitis." These inflammations are expressed as follow:—(1.) *Simple erythema;* (2.) Diptheritic exudation or eruption; (3.) Inflammation of the follicles of the mucous membrane—*follicular stomatitis.*

Simple Erythema occurs in patches, and is generally due to hot or acrid substances taken into the mouth, to cold, or the irritation of the teeth, or of tartar upon them. It may be also due to gastric derangement. Small doses of *magnesia*, or of *rhubarb with soda*, will generally correct the stomach derangement, when the stomatitis will subside.

Diphtheria of the Mouth—Thrush, or *Muguet*—is a pultaceous product of inflammation, in which the secretion of the mouth is greatly altered and increased. The disease chiefly attacks the new-born infant. The whole surface of the mouth exhibits unusual redness, with here and there cord-like exudation in irregular patches, preceded by vesicles, especially behind the lips and about the tip of the tongue. These patches are thrown off, to be renewed again; and the mucous membrane below is of a bright red colour. The adjacent glands are apt to become tumid and tender. The skin is commonly hot and dry; thirst is considerable; swallowing seems to give pain; and diarrhœa may ensue to a degree which soon proves fatal in an infant. The disease frequently occurs as a sequela of measles.

Treatment.—The mouth should be frequently washed with emollient fluids, such as *lintseed infusion, diluted glycerine*, and *biborate of soda*, or *honey* mixed with *biborate of soda.*

Dr. Tanner recommends the following lotion:—

℞. Sodæ Biboratis, ʒi.; Glycerinii, ʒii.; Aquæ Rosæ, ʒiv.; *misce.* To be painted over the lips and mucous membrane of the mouth and tongue.

Creosote, vinegar, carbolic acid, glycerine, and *alcohol* are also recommended as local applications. In severe cases, where the breath becomes fetid, with the submaxillary glands large and swollen, the lips and gums tumid, the face flushed and swollen, and the fever intense, *chlorate of potash* must be freely given, to the extent of *five* grains every four or six hours.

Follicular Inflammation of the Mouth, Follicular Stomatitis, Aphthous Stomatitis or *Aphthæ of the Mouth.*—This disease usually commences as a simple stomatitis; but very soon small, round,

transparent, greyish or white vesicles appear, and at the base of each is an elevated marginal ring, which is pale and firm. Fluid soon escapes from the ruptured vesicle; an ulcer forms, which spreads, bounded by a red circle and an elevated border. In some forms of the affection microscopical parasitic plants occur.

Treatment.—Besides the remedies already recommended, it will be found that the solution of the *pernitrate* of iron, internally, has a beneficial effect upon the sores. It is prescribed in the following formula (DUNGLISON):—

℞. Liq. Ferri Pernitratis, gtt. xl.; Syrup. Aurant., ℥ss.; Aquæ, ℥vss.; *misce.* A fourth part may be given to a child three or four years of age four times a day.

In cases where parasitic vegetable productions abound, the application of a solution of *sulphate of soda* (℥i. to ℥i. of water) removes the lesion in twenty-four hours (JENNER). The secretions of the mouth being acid, the salt is decomposed, and sulphurous acid is set free, which destroys the parasite. A change of air is often absolutely necessary to restore the patient to health; and arsenic and iodine are useful restorative agents in repairing, with good diet, the faulty nutrition of the child.

Ulcerative and Gangrenous Inflammation of the Mouth, or *Noma and Cancrum oris.*—These are but different grades of similar lesions. The *noma,* or milder form, generally commences at the edges of the gums, opposite the incisors of the lower jaw. At these points the gums appear white, become spongy, and separate from the teeth, as if mercury had produced its specific effects. Ulceration begins, and extends along the gums until the jaws are implicated; and as the disease advances, the cheeks and lips begin to swell, so as to form a tense indurated tumefaction. The teeth are apt to fall out; and the gums assuming a gangrenous condition, the breath becomes intolerably fetid. There is generally enlargement with tenderness of the submaxillary glands.

The more severe form (*cancrum oris*) occurs in children of debilitated habits, between *two* and *five* years of age especially. A copious flow of saliva, a tumid appearance of a cheek, with fetor of breath, ought to suggest an examination of the mouth when small vesicles of a greyish-red or even black appearance may be seen on the inside of the lips or the tumid cheek. These vesicles are surrounded by a red base; and swelling, surrounding

hardness, heat, and pain increase. An ash-coloured eschar may then appear in the centre of the cheek, within the cavity of the mouth, surrounded by a glossy tumefaction of the parts; and on the inside of the cheek a hard indolent swelling. So rapid may be the spread of this destructive disease, that in a few days the lips, cheeks, tonsils, palate, tongue, and even half the face, may become gangrenous, the teeth falling from their sockets, a horribly fetid saliva and fluid flowing from the parts.

Treatment.—Analogous to hospital gangrene, an early recognition of the nature of the disease will suggest an efficient application of *strong nitric acid* to the slough. The mouth must be frequently syringed with a solution of *carbolic acid,* in the proportion of *half a drachm dissolved in a gallon of boiling water, and allowed to become warm or tepid.* Tonics and antiseptics must be freely given. *Beef tea, wine, brandy, quinine,* and *chlorate of potash,* are the remedies indicated.

GLOSSITIS.

LATIN EQ., *Glossitis;* FRENCH EQ., *Glossite;* GERMAN EQ., *Zungenentzündung;* ITALIAN EQ., *Glossitide.*

Definition.—*Inflammation of the tongue.*

Pathology.—*The tongue* is liable to acute inflammation of its substance generally, to various forms of ulcers, and to cancer.

Inflammation of the Tongue, or *Glossitis,* may result from acrid substances taken into the mouth, or from the specific action of *mercury,* or *scarlet fever,* or *small-pox.* There is generally great tumefaction from infiltration of serum; while fever, mental depression, and general weakness prevail, with pain and heat of the tongue, the colour of which is of a deeper red than usual. The swelling may be so great as to cause the tongue to project beyond the teeth, or even to project so far back as to cause dyspnœa. Dr. Graves relates a case of inflammation affecting one-half the tongue, the median line forming the boundary between the swollen and the healthy parts.

Treatment.—Active cathartics are generally of great service; and they are to be given as enemata. Blood must be taken directly from the tongue in such cases. Incisions along the inflamed organ, followed by the action of the vapour of hot

water, may reduce the swelling and relieve congestion. In Dr. Graves's case, two or three applications of six leeches at a time to the inflamed parts produced a speedy decrease of the tumor. If suffocation is imminent, tracheotomy or laryngotomy may be performed.

Ulcers of the Tongue are of various kinds:—(1.) Simple ulcers are usually situated about the tip, near the frænum, or at the edges; (2.) Ulcers resulting from the action of mercury are usually associated with similar ulcerations of the gums and mercurial fetor of the breath; (3.) Syphilitic ulcers and eruptions are generally superficial—often presenting (*a.*) A glossy *eczematous* or psoriasis-like patch, which creeps from place to place, and which heals and breaks out again repeatedly. (*b.*) Sometimes an inelastic induration or node will form on the upper back part of the tongue, often in such a position facing the epiglottis that the mirror or speculum is absolutely necessary to see the lesion. (Several such lesions are to be seen in the Museum of the Army Medical Department.) These nodes generally commence to slough in the centre, having ragged, thickened, and hard edges. (*c.*) Other forms of syphilitic lesion of the tongue are seen in long fissures or cracks, with enormous hypertrophy of the papillæ along the edges of the fissure, forming warty condylomatous excrescences. Primary chancres sometimes exist.

Treatment.—Simple ulcers soon heal under the influence of water simply, or borax solutions frequently taken into the mouth as a gargle. Ulcers from mercury heal best under the use of *sulphate of copper* or other astringent gargles. Syphilitic nodes of the tongue may yield to calomel and opium, or to opium and grey powder, in small doses every night—constitutional treatment by diet being attended to at the same time.

TONSILLITIS.

LATIN EQ., *Inflammatio tonsillarum;* FRENCH EQ., *Amygdalite;* GERMAN EQ., *Entzündung der Mandeln;* ITALIAN EQ., *Tonsillitide.*

Definition.—*Inflammation of the tonsils.*

Pathology.—This disease is indicated by redness of the tonsils, which are loaded with blood, and moderately swollen. The *uvula* generally participates in the morbid action. It becomes

elongated, and rests on the base of the tongue, causing a most disagreeable sense of titillation. Lymph or serum may be thrown out, and in this case the tonsils are often greatly swollen, so as in some instances almost to block up the passage of the fauces. The diagnostic symptom between the effusion of serum and of lymph is that, in the latter case, the tonsil remains often permanently enlarged. In a few instances an abscess forms in the tonsils, which ultimately ruptures, and discharges a greater or less quantity of pus. It generally happens that both tonsils are affected, but occasionally the inflammation is limited to one.

In chronic inflammation of the tonsil the same phenomena are seen, but the course of the disease is slower, and the colour of the parts less vivid, and in general differing little from their natural tint. The distended gland-vesicles, filled with their secretion, distend the follicles, so that the tonsils appear dotted over with little abscesses. These gland-cavities eventually rupture, and discharge more or less consistent cheesy-like pellets, having an excessive fetid, fæcal-like odour.

The inflammation of the fauces, whether acute or chronic, not unfrequently extends to the pharynx. The inflammation may commence in the subcellular pharyngeal membrane, and an abscess occasionally forms in that part. In a smaller number of cases, by an extension of the original disease, the *epiglottis*, *glottis*, and even the *larynx* are affected. In bad cases, as after severe scarlatina, it may spread to the Eustachian tube, and cause suppurative or other inflammation of the mucous membrane of the internal ear. It sometimes extends up the nasal passages, by which respiration through those passages is much impeded or rendered impossible, so that the patient breathes with his mouth open.

Symptoms.—The different degrees of intensity which attend this affection have suggested a division of sore throat into *angina mitior* and into *angina gravior*.

Angina gravior is usually preceded by some shivering and fever, which having lasted a few hours, the patient has the sensation of a sore throat. He finds deglutition difficult and painful, and what he attempts to swallow is perhaps rejected through the nostrils; his voice is altered, being hoarse and nasal, and he can hardly breathe through his nose; his ears are also painful, and he finds it troublesome to free his throat

from the viscid matters which adhere to it. Fever does not abate on the appearance of the local affections, but continues till the abscess of the tonsil bursts, or till resolution is complete. The degree of prostration which attends *tonsillitis* is usually out of all proportion to the severity of the local lesion. It generally subsides in three or four days, and rarely lasts longer than a week. To inspect the throat well, the patient should be directed to open his mouth widely. The dorsum of the tongue must then be depressed by some flat body, such as the handle of a spoon, the patient being made to inhale a full breath. The tonsils can then generally be seen without much difficulty.

Treatment.—In the early stage cathartics are very useful, with very hot foot baths, containing mustard. Emollient gargles, with the inhalation of steam, should be applied to the throat. Dover's powder, to the extent of three grains every four or six hours, with warm diluents, often greatly subdue the febrile state; but the effects of the opium must be watched, so as not to be carried too far. The gum resin of *guaiacum*, in the following formula, is most efficient in subduing incipient *tonsillitis*:—

℞. Magnes. Sulph., ℥vi.; solve in Aqua, ℥viii.; adde Pulv. Guaiac., ℥iss.; Pulv. g. Tragacanth. Co., ℥ii.; *misce bene*. A sixth part every four hours till the bowels are freely opened.

When the inflammation advances to suppuration, it may be necessary to open the abscess; but generally it bursts of itself, during some effort of the parts, in deglutition. If a puncture is necessary, its direction must be towards the middle line.

PHARYNGITIS.

LATIN EQ., *Pharyngitis;* FRENCH EQ., *Pharyngite;* GERMAN EQ., *Schlundentzündung;* ITALIAN EQ., *Faringitide.*

Definition.—*Follicular inflammation of the pharynx.*

Pathology—Is indicated by huskiness of the voice, with more or less coughing, hawking, or spitting. The follicles of the *isthmus faucium* and of the pharynx are unusually apparent; and the mucous membrane seems studded with granulations, probably due to an accumulation of secretion in the follicles. Occasionally the follicles burst, and discharge small masses, of an elastic consistent nature, causing great anxiety to nervous patients. Ulcer-

ation may then supervene (GREEN, DUNGLISON). The patient at first feels an uneasy sensation in the throat, with a constant inclination to swallow, as if an obstacle existed in the œsophagus. It is a disease apt to occur amongst clergymen, and men who have much speaking in public, and has been known as "clergyman's sore throat."

Treatment.—Tonics, such as *iron*, *quinine*, and *strychnia*, are mainly indicated. The local applications most useful are solutions of *nitrate of silver*, or *creosote* solution. The nitrate of silver is used in the proportion of *two to four scruples to the ounce* of water, applied by a *probang*, about ten inches long, to the affected mucous surfaces. Time is a most important element in the cure; for the disease is essentially chronic in character.

The treatment of diseases of the *pharynx* and of the *larynx* by the use of *atomized fluids*, in which medicated vapour and gases are brought to act upon the parts, has recently been used with advantage, since better appliances have been devised for using such remedial agents. The aim of such instruments is to atomize or pulverize, or very minutely to divide the fluid, by causing it to be thrown as a fine shower or spray, so as to be inhaled as such by the patient.

Dr. Andrew Clark's hand-balls (fig. 42), fitted with Bergoon's tubes and Maunder's atomizer, constitute an efficient and useful

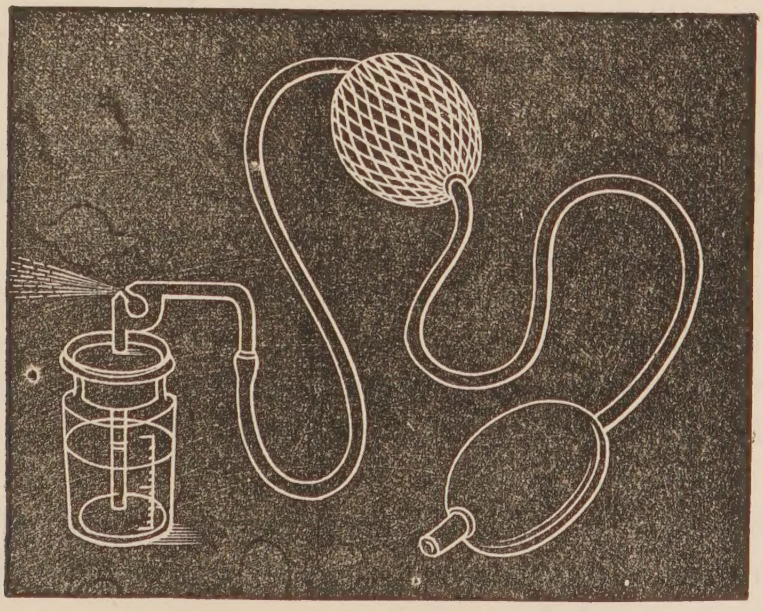

Fig. 42.

arrangement for applying fluids in this way up the nostrils, to the back of the throat, or over the entrance to the larynx (DA COSTA).

Dr. Richardson's well-known instrument (fig. 43), for the production of local anæsthesia is also an excellent atomizer of fluid, and may be used for a similar purpose.

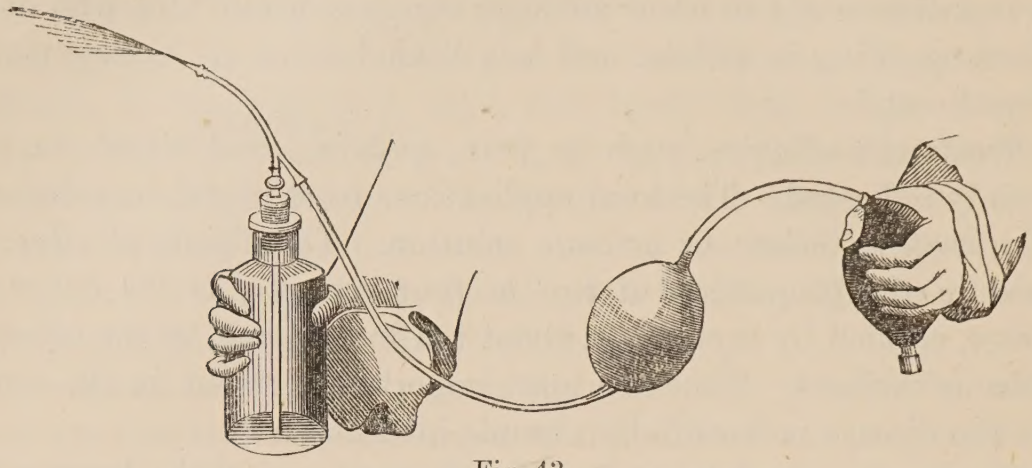

Fig. 43.

Where it is desirable to make the topical application directly upon the diseased part, these are the most simple instruments that can be used without loss of time by the physician; but where *inhalation* simply of vapour or heated air variously medicated is all that is required, nothing can be better than that described at p. 666, used by Dr. Fergus, of Glasgow.*

On this most useful mode of treatment Dr. Da Costa makes the following observations:—

"Inhalations by means of atomized fluids are an unquestionable addition to our therapeutic means; but they are nothing but an addition, and not a substitute for all other treatment.

"In most acute diseases of the larynx, and still more so in acute disorders of the lungs, their value, save in so far as those of water may tend to relieve the sense of distress, &c., and aid expectoration, is very doubtful; though in some acute affections, such as in œdema of the glottis and in croup, medicated inhalations have strong claims to consideration.

"In certain chronic morbid states of the larynx, particularly those of a catarrhal kind, and in chronic bronchitis, they have proved themselves of great value.

"In the earlier stages of phthisis, too, they may be of decided advantage; and at any stage they may be a valuable aid in treating the symptoms of this malady.

"Their influence on such affections as hooping-cough and asthma is not satisfactorily proved.

* It is furnished by Mr. H. Reid, Chemist, 423 Argyle Street, Glasgow.

"They furnish a decided and unexpected augmentation of our resources in the treatment of pulmonary hæmorrhage.

"They require care in their employ; and in acute affections we should consider whether, as they have to be used frequently to be of service, the patient's strength justifies the disturbance or the annoyance their frequent use may cause.

"In any case, to be of service, they ought to be carried on as a treatment with a distinct object, and not intermittingly or spasmodically resorted to."*

* Table of Doses for Inhalation (Da Costa)—Alum, 10 to 20 Grains.—In this dose, suitable to chronic catarrhal affections of pharynx and air-tubes, particularly in bronchial affections with excessive secretion, when, as in most inflammatory conditions of the respiratory mucous membrane, it may be advantageously united with opium. In rather larger doses, 30 grains to the ounce, useful in pulmonary hæmorrhage. Is, as an astringent, generally more of a sedative, and more suited to conditions of irritation than tannin (Fieber).

Tannin, 1 to 20 Grains.—Useful for the same affections as alum. Employed in cases of laryngeal ulceration and excrescences, in œdema of the glottis (Trousseau), in croup. Here, as well as in pulmonary hæmorrhages, in large doses. In ordinary cases of laryngeal or bronchial disease, begin with a small dose. If the remedy occasion much heat and dryness, it is not to be employed.

Iron (perchloride of), $\frac{1}{8}$ to 2 Grains.—In earlier stages of phthisis. In chronic pharyngitis or laryngitis may be used stronger. As a weak inhalation in hysterical aphonia. Of greatest strength in pulmonary hæmorrhage, 2 to 10 grains to the ounce. The lactate, citrate, or phosphate may, in ordinary cases in which we wish a non-astringent salt of iron, be also used, though they are not, on the whole, as available as the chloride.

Nitrate of Silver, 1 to 10 Grains.—In ulcerations of pharynx and larynx, in follicular pharyngitis. A face-shield is always to be used. Ten grains to the ounce only in cases of ulceration.

Sulphate of Zinc, 1 to 6 Grains.—In bronchial catarrh with excessive secretion. In aphonia connected with chronic laryngeal catarrh.

Chloride of Sodium, 5 to 20 Grains.—Promotes expectoration and diminishes sputa; employed in phthisis.

Chlorinated Soda (*Liquor Sodæ Chlorinatæ*), $\frac{1}{2}$ to 1 Drachm.—In bronchitis with offensive and copious expectoration; in phthisis.

Muriate of Ammonia, 10 to 20 Grains.—In laryngeal and bronchial catarrh, acute as well as chronic. To promote expectoration; also in capillary bronchitis. The dose best borne is not above 10 grains to the ounce.

Opium (watery extract of), $\frac{1}{4}$ to $\frac{1}{2}$ a Grain.—In irritative coughs, and as an adjunct to allay irritation. Also for its constitutional effects. Dose of tincture of opium, 3 to 10 drops. Acetate of morphia, one-twelfth to one-eighth of a grain, has been administered; but large doses require much caution.

Conium (fluid extract of), 3 to 8 Minims.—Irritative cough; asthma; feeling of irritation in larynx.

Hyoscyamus (fluid extract of), 3 to 10 Minims.—Spasmodic coughs; hooping-

LARYNGITIS.

LATIN EQ., *Laryngitis;* FRENCH EQ., *Laryngite;* GERMAN EQ., *Entzündung des Kehlkopfs;* ITALIAN EQ., *Laringitide.*

Definition.—*Inflammation of the larynx.*

Pathology.—It is from the situation of the lesion that affections of the larynx derive their importance, their gravity, and their great danger. *Acute laryngitis* is not only dangerous to life, but there are few diseases which can kill quicker. Death approaches by suffocation. The width of an eighth of an inch or so in the chink of the *glottis* may be all the passage left to breathe by; and the œdema which attends the disease may so completely close the chink, that death may ensue in two or three minutes.

It is the œdema which attends the inflammation which makes laryngitis so dangerous a lesion. The disease may supervene with rigors like a common cold; but very soon a sense of constriction and strangulation occurs about the throat; and the voice becomes affected, or completely disappears. The most striking phenomena are expressed by modifications of the natural functions of the part. Vocalization, cough, and respiration are all more or less modified. While the patient breathes he makes a noise in his throat, both during expiration and inspiration. The noise is like the sound of a whisper, and may be heard at a considerable distance. Inspiration is laboured and tedious, and starts sharp from the conclusion of the previous expiration; and both are accompanied by a rasping, hissing sound. Voice is gone, which shows that the *larynx* is the part affected, because

cough. One-half grain of the extract, gradually increased, or the tincture may be employed.

CANNABIS INDICA (tincture of), 5 TO 10 MINIMS.—In spasmodic coughs; phthisis.

IODINE (*Liq. Iodinii Compos.*), 2 TO 15 MINIMS.—In chronic bronchitis; in phthisis.

ARSENIC (*Liq. Potass. Arsenit.*), 1 TO 20 MINIMS.—Nervous asthma (TROUSSEAU).

TAR WATER, 1 TO 2 DRACHMS of officinal solution.—In offensive secretions from bronchial tubes; in tuberculosis; as an antiseptic in gangrene of lungs.

TURPENTINE, 1 TO 2 MINIMS.—In chronic bronchitis with offensive secretions; in bronchorrhœa; in gangrene of lungs.

LIME WATER, used of officinal strength, or stronger.—In diphtheria; in membranous croup.

WATER, Distilled.—Cold, in pulmonary hæmorrhage. Warm water in asthma; in croup; in bronchitis.

there, and there only, voice is formed. The cough of disease of the larynx is also peculiar. It is brassy in its tone, terminates in a hissing noise, and begins similarly by a hissing inspiration in a muffled manner, because the lips of the glottis being thickened, irregular, and rough, cannot be sufficiently closed to begin a sharp sound (HYDE SALTER). Laryngitis is one of the legitimate results of a catarrh, and if once it occurs is liable to occur again.

Treatment.—If there be time, commence by the inhalation of the steam of boiling water, and continue to breathe hot moist air, as much by the nostrils as possible. Leeches may also be applied to the throat, followed by hot fomentations. If benefit do not follow these remedial measures, tracheotomy ought not to be delayed. The air of respiration ought then to be warm, moist, and plenty of it, through a large-sized canula, the orifice of which must be kept free of secretion.

LOSS OF VOICE—SYN., APHONIA.

Definition.—*When the larynx is so affected that the voice is wholly or partially lost, the patient is unable to speak except in a whisper.*

Pathology.—Whatever affects the muscles of the larynx, as any overstraining of the voice in singing or speaking, cold, or sudden changes of the weather, is apt to produce a more or less temporary loss of voice. Rheumatic affection of those parts is also a cause, as likewise all that impairs the nervous energy of the laryngeal muscles. Thus the voice is often lost after a paroxysm of hysteria, or a severe mental affection. It is well known, also, that at the period of menstruation, two or more of the upper notes of the voice are apt to be lost for the time. Cases of dilated aorta, or of aneurism of the aorta about its arch, are apt to be associated with temporary aphonia.

Complete aphonia is common to all ages, but is most common in early adult age, and more particularly in the female. In advanced age the change of the voice, and the general impairment of its tone and volume, are well known. Many singers who have compassed two octaves in their prime hardly, perhaps, preserve four feeble notes in old age. This, perhaps, among other changes, may be owing to ossification of the cartilages.

Tumors and warty-like growths have been recognized by the

laryngoscope (see p. 795, *ante*, fig. 37); but an entire loss of the voice often takes place without any congestion, inflammation, or other structural lesion of the tissues of the larynx and glottis. When aphonia is secondary or symptomatic, tubercular or other structural diseases of the lungs are often found.

Symptoms.—In primary aphonia there is no tenderness or soreness of the larynx, no pain on pressure, and no expectoration, and the general health of the patient is good. It often comes on suddenly, and only in a few instances is the attack gradual. It often also disappears in a few hours, but in other cases it continues for some weeks, and even months.

Diagnosis.—Aphonia is so marked a symptom, that though some doubt may exist as to the cause, none can exist as to the disease. It is distinguished from the aphonia in phthisis, or other malignant diseases, by the general good health of the patient; and the laryngoscope ought to be used to see the condition of the vocal cords.

Prognosis.—Primary aphonia is seldom of any moment. When it results from phthisis, it is one of those symptoms which mark the tendency of the disease to a speedy fatal termination.

Treatment.—Aphonia is often very difficult to cure. Sometimes attention to the general health will remove it. In other cases it yields to some local application, as blisters, mustard poultices, or the camphor liniment, or other similar stimulating applications. Tonic regimen, the fundamental basis of which may be summed up in the three words, "air, exercise, and diet," of the most sanitary kind, is generally sufficient to bring about the cure. The shower-bath has often been of service; and it should be remembered that this disease can be easily feigned.

INFLAMMATION OF THE MUCOUS MEMBRANE OF THE ŒSOPHAGUS.

Œsophagitis is a rare disease, for morbid poisons seem to have little influence over this portion of the alimentary canal, and atmospheric vicissitudes are in like manner seldom followed by inflammatory affections of this part. The most frequent causes of inflammation of the œsophagus are, accidentally drinking boiling water; swallowing corrosive liquids, as the mineral acids; and wounds, most commonly inflicted in the act of committing suicide.

Children a few days old are sometimes affected with slight inflammatory affections of the œsophagus.

Inflammation of the mucous membrane of the œsophagus is characterized by a deep redness of the part, generally terminating by resolution, but occasionally followed by separation of the cuticle. Lymph may be thrown out. In new-born children points of lymph are often found lying on the mucous membrane of the œsophagus, being apparently an extension of the thrush affecting the mouth and pharynx. Andral has seen, in a girl twelve years old, lymph thrown out after the manner of broad bands in the pharynx, œsophagus, and stomach. After puberty this form of inflammation is still more rare, but there are some few instances. Cruveilhier says that he found among the preparations of Dupuytren a very remarkable example of inflammation of the œsophagus, terminating in the formation of a false membrane, which coated this canal throughout its whole length. Dr. Abercrombie also gives the case of a gentleman, aged twenty-six, who caught cold, and died in about three weeks. The whole of the pharynx was covered by a loose adventitious membrane, which extended over the epiglottis, and portions of it were found lying in small irregular masses within the larynx at the upper part. A similar membrane was traced through the whole extent of the inner surface of the œsophagus quite to the cardiac orifice.

Besides lymph being thrown out, the mucous membrane of the œsophagus may also ulcerate, especially as a result of irritant poisons. These ulcers in general form on the anterior portion of the œsophagus; and by continued extension they at last penetrate the posterior surface of the larynx, so that the patient often dies suffocated from the escape of food into the lungs. Occasionally the ulceration takes place from without inwards. The cicatrices are very apt to induce stricture. In cases of poisoning with the mineral acids, the whole œsophageal canal may become constricted and narrowed, and the mucous membrane puckered up and contracted, so as greatly to diminish the calibre of the canal generally. More commonly the stricture is partial, one circular muscular fibre perhaps having been abnormally contracted, and in this state bound down by adhesive inflammation, diminishing the diameter of the canal at that part to at least one-half. Dr. Baillie mentions a case in which, from this cause, the diameter of

the œsophagus was so reduced as hardly to allow a garden pea to pass; yet in all other respects the œsophagus was healthy.

Symptoms.—The symptoms of *œsophagitis* are almost entirely local, and consist principally of pain, of dysphagia, of the expectoration of a thick viscid mucus, and perhaps of vomiting. Emaciation follows the loss of nutrition, and the patient ultimately dies from inanition. Stricture may be induced by the careless introduction of a probang.

Diagnosis.—The diseases with which it may be confounded are similar states of the stomach; and the diagnosis in these cases is often difficult and perplexing. Stricture may be confounded with the spasmodic affections caused by an irritated state of the lung or trachea.

Prognosis.—Simple *œsophagitis* is probably often recovered from, as is seen after wounds of the throat partially dividing the œsophagus; but the chronic forms of inflammation probably often lay the foundation of disease leading to the ultimate death of the patient. Ulceration extending into the thoracic or pericardial cavity has been in all cases fatal.

Treatment.—The treatment of *œsophagitis* is by small local bleedings, by warm cataplasms to the neck, and by moderately acting on the bowels. In the treatment of the more chronic forms some sedative is essential. The use of the probang must be left to the discretion of the practitioner. There is always some danger, in its use, of rupturing the canal, or of causing an ulcer. When the case is hopeless, from the small quantity of aliment which reaches the stomach, life may yet be prolonged by enemata of soups, milk, egg wine, or other nutritious fluid matters.

CHAPTER XII.

ON THE DIAGNOSIS OF BOWEL DISEASES.

Section I.—Relation of the Abdominal Viscera to the Walls of the Abdomen.

The regions into which the abdomen is usually divided by lines are, like those of the thorax already described, quite arbitrary; and figs. 5, 6, 7, already given, are referred to here, likewise the

description given at p. 524, as to the fixed skeleton points which determine the direction of these lines, and the regions mapped out by them.

The vertical lines which have reference to the abdomen are five in number, and run as follows:—(1.) From the insertion of Poupart's ligament in the external tubercle of the pubes to the acromial extremity of the clavicle (right and left side); (2.) From the posterior boundary of the axilla (the inferior edge of *latissimus dorsi*) to that point of the crest of the ilium on which it falls vertically, fig. 6 (right and left side); (3.) Along the spinous processes of the vertebræ from the sacrum to the nape of the neck.

The transverse lines are four in number, and indicate horizontal planes as follow:—(1.) On the point of the xyphoid cartilage; (2.) On the last short ribs; (3.) On the anterior superior spinous process of the ilium on each side; (4.) On the upper margin of the *os pubis*. These three horizontal and five vertical bands map out the abdominal walls into *thirteen* regions, of which five are anterior, four are lateral (two on each side), and four are posterior. They are named as follows:—*Anterior regions* are *epigastric* (No. 4 on the figures); *umbilical* (No. 5); *hypogastric* (No. 6); right and left *inguinal* (No. 11). The *lateral regions* comprehend the right and left *hypochondriac* (No. 9); the right and left iliac (No. 10). The *posterior regions* embrace the *inferior dorsal* on the right and left (No. 15); the right and left *lumbar* (No. 16).

The best idea of the contents of these regions is obtained by defining first the limits of the *liver*. It fills the right *hypochondriac* region (No. 9, figs. 5, 6), filling up the concavity of the diaphragm; and it is almost completely concealed by the arch of the ribs. A part of the left lobe projects into the *epigastric* region (No. 4, figs. 5, 6) and *left hypochondriac*. It also projects upwards into the *infra-axillary* region (No. 8, figs. 5, 6) of the thorax, where it is separated from the thoracic wall by the thin lower margin of the right lung. Its upper margin in this space is on a line nearly with the level of the nipple, about the fifth intercostal space—less frequently beneath the fifth rib. In the perpendicular axillary line its margin is about the *seventh* intercostal space—more seldom under the seventh rib: close to the vertebral column its margin is in the tenth intercostal space—less

frequently in the ninth (FRERICHS). At the median line the upper boundary of the liver cannot usually be distinguished from the lower margin of the heart. It is best made out by drawing a straight line from the point of contact of the right margin of the cardiac dulness with the upper boundary of the liver to the apex of the cardiac dulness on the left (CONRADI, FRERICHS, vol. i., p. 30).

Percussion of the liver after a meal is to be avoided; and any obstinate constipation which may be present must be removed before percussion, by means of free purgation, and any accumulations of gas must be got rid of.

In the epigastric and left hypochondriac regions lies the stomach. The umbilical region is crossed by the transverse colon, passing from right to left a little above the umbilicus.

The convolutions of the jejunum and ileum occupy the umbilical and hypogastric regions. The large intestines surrounding the convolutions of the lesser intestines occupies the iliac and lumbar regions on each side. The kidneys are equally shared between the *infra-scapular* (No. 14, fig. 9) and the inferior dorsal regions (No. 15, fig. 9). The spleen in its greater bulk is in the same region on the left side.

SECTION II.—METHODS OF EXPLORING THE ABDOMEN.

These are principally three—namely, *inspection, manual examination* (*palpation*), and *percussion.*

Inspection furnishes information relative to *size, form,* and *movement;* and such information ought always to be acquired when the chest, as well as the abdomen, are both exposed simultaneously in a good light, the patient being protected from cold by a previous regulation of temperature in a room suited for the purpose of such an examination. The eyes of the patient ought to be directed away from the examiner.

Palpation furnishes information relative to position, size, consistence, elasticity, spontaneous movement, or mobility, and the presence of vibrations which may reach the surface. Care should be taken that the hand, when applied, should not be cold.

Percussion furnishes information relative to the comparative solidity of regions, and thereby indicates the kind of organ immediately below the seat of percussion.

The chief objects to be held in view in exploration of the abdomen are the following:—

To ascertain—(1.) Its form and size; (2.) Its degree of tension or solidity; (3.) Its temperature; (4.) Sensibility or tenderness over any part; (5.) The presence or absence of tumor in or amongst the viscera; (6.) The presence or absence of fluids in the peritoneal sac; (7.) The nature and extent of the intestinal contents.

CHAPTER XIII.

DISEASES DURING WHICH LESIONS TEND TO BE LOCALIZED IN THE ABDOMINAL CAVITY AND CONTAINED VISCERA.

SECTION I.—DISEASES ASSOCIATED WITH ORGANIC LESION OR FUNCTIONAL DISORDER OF THE PERITONEUM.

PERITONITIS.

LATIN EQ., *Peritonitis;* FRENCH EQ., *Péritonite;* GERMAN EQ., *Bauchfellentzündung;* ITALIAN EQ., *Peritonitide.*

Definition.—*An inflammation of the serous membrane lining the cavity of the abdomen, and covering the viscera contained in that cavity.*

Pathology.—The peritoneum, like the pleura, is liable to inflammatory processes, either acute or chronic in their progress.

Acute inflammation of the peritoneum (as of all serous membranes) begins in the connecting fibrous tissue, which becomes red and injected, and at length the same phenomena pervade the serous membrane itself. Its colour when inflamed is a bright arterial scarlet hue—being first dotted with a number of small red points, which become confluent, and form streaks and patches, which in their turn coalesce; or a small central nucleus of inflammation may form, and spread till the whole extent of the peritoneum is one entire bright red colour. In addition to the redness, some interstitial growth or exudation accompanies diffuse inflammation of the peritoneum, so that the membrane loses its transparency, and is thickened. When redness does not

exist, opacity is often the only evidence of the previous existence of the inflammatory state. The consistence also of the subperitoneal tissue is greatly impaired, and rendered easily lacerable, so that the peritoneum is capable of being detached in considerable portions. This inflammation may terminate by resolution, or it may advance to the effusion of serum. The quantity may be trifling, not exceeding a few ounces, but occasionally it is large, fills the cavity of the abdomen, and constitutes inflammatory dropsy.

The exudation may be of the fibrinous type, when coagulation of the effused fluid tends to occur, and the opposed surfaces to be glued together. In some cases, however, the fluid predominates, and the fibrinous coagulated masses are loose, so as to float unattached in the serum, or of such consistence as to unite opposite parts together, and of such extent as sometimes to form an adventitious membrane, covering the entire surface of the abdominal walls as well as the whole of the intestines. The period at which organization of the lymph thus effused may begin, Mr. Hunter determined to be about twenty-four hours. If the disease proceeds, pus forms, sometimes not to a greater amount than a few ounces; but in other cases it amounts to many pints, or even fills the whole of the abdominal cavity. Ulceration of the peritoneum is not frequent, and generally takes place from without inwards, as from a perforating ulcer of the small or large intestines, or from the rupturing of an abscess or other tumor. The different acute inflammations described have been mentioned as though succeeding each other; but in many instances all these different forms co-exist in different parts of the peritoneum at the same time, and perhaps have been irregularly set up.

Experience has also shown that although the structure of the peritoneum appears to be uniformly the same, yet certain parts of it are more liable to inflammation than others, as the convex surface of the liver or spleen, the right iliac fossa, the surface of the small intestine, and in females the broad ligaments, the Fallopian tubes, and the parts immediately adjoining them, as also the space covering the rectum and bladder. The parts the most rarely affected are those covering the stomach, bladder, omentum, and the mesentery. It will be seen that the liability of different parts of the peritoneum to inflammation is in proportion to the liability of the organs they cover to become diseased, and that

these partial inflammations are for the most part the result of contiguous irritation.

The forms of peritonitis to be distinguished are,—(a.) *Puerperal peritonitis;* (b.) *Chronic peritonitis;* (c.) *Suppurative peritonitis;* (d.) *Tubercular peritonitis;* (e.) *Adhesive peritonitis,* and local adhesions of the parts opposed to each other.

Symptoms.—Peritonitis may be acute or chronic, partial or general. It is occasionally ushered in by some previous shivering and fever, but in many cases there are no preliminary symptoms. The symptoms of peritonitis from perforation of the intestine or stomach are characterized by the *suddenness* and *intensity* of pain, often referred to a particular region of the abdomen; but the whole abdomen soon becomes painful to pressure. The pain is constant, exquisite, and leads to such lowering of the heart's action that death rapidly follows by *asthenia,* preceded by the most marked symptoms of collapse.

If acute peritonitis should not terminate by resolution, but by effusion of serum or of lymph, the patient complains of a severe pain in the abdomen, which is increased on pressure. He lies on his back, fearing to move. His pulse is from 90 to 120, and peculiar, as an inflammatory pulse. In proportion as it is frequent, so is it smaller. The tongue is coated, and the bowels constipated, or regular. If serum be effused, that event can often be determined by fluctuation, or by percussion in some parts; or if lymph, by a rubbing sound heard under the stethoscope when the abdominal movements of respiration are not suppressed. The course of these forms of acute peritonitis varies from a few hours to ten or fourteen days.

When acute peritonitis terminates in effusion of pus, the symptoms are infinitely more formidable. The pain in the abdomen is often the severest that human nature can suffer. The patient lies on his back, but his legs are drawn up and bent so as to relax as much as possible the abdominal muscles. By fixing his pelvis he endeavours to keep the abdomen still; he is restless, and unable to bear the slightest pressure, not even the weight of a sheet, and is incessantly tossing his arms about in every direction. The state of his tongue and bowels is similar, perhaps, to what has been described; but his pulse is excessively small and rapid, varying from 130 to 150, while the patient is often distressingly affected by retching and vomiting. These symptoms per-

haps continue without intermission for twenty-four, forty-eight, seventy-two, or more hours; when, with or without some previous shivering, pus is effused, and the pain, from being agonizing, is now bearable. The subsidence of the pain, however, is not followed by any amendment; on the contrary, a most alarming collapse succeeds,—a cold, clammy sweat breaks out over the body, while hiccup, and a pulse hourly increasing in frequency, proclaim the entire hopelessness of the patient's surviving beyond a few hours.

When acute peritonitis is local—confined, for instance, to the surface of the liver or other organ—the pain is often limited to that part, while the other symptoms vary according to the severity of the affection and the organ whose covering is affected.

Chronic peritonitis often takes place to a great extent, and yet without any great amount of suffering. The symptoms are rather those of abdominal soreness and uneasiness than of pain, together with a full but sometimes rapid pulse. The intestines, indeed, may be glued together, and sometimes pus has been found effused, without the patient suffering more than in ascites. When chronic peritonitis is partial, as of the liver or spleen, the patient often experiences a dragging pain, which is increased by a change of position, and arises from the parts being suspended by adhesion.

Causes.—Inflammation of the peritoneum often manifests itself during the course of some specific disease, such as paludal fevers, scarlet fever, and the like. Mechanical violence, as the kick of a horse, the operation for hernia, ovariotomy, or the stone, or of paracentesis, are occasional causes. Rupture of the intestine from ulceration, or the bursting of an abscess, or of an aneurismal tumor into the abdominal cavity, are examples of another class of causes. Errors of diet, and especially frequent intoxication, are also occasional causes. The disease termed *gin colic* is a chronic inflammation of the peritoneum. Sudden and great changes of temperature are also causes, especially in women at the period of menstruation. Intus-susception of the intestine, or strangulation of the intestine from hernia, or other accidents, are also occasional causes. As a secondary disease it is frequently produced by hepatitis, splenitis, enteritis, and by *cancerous*, *typhoid*, and *tubercular* deposits in the subperitoneal tissue.

Children sometimes die of this affection after fevers. Peri-

tonitis, however, is most common between the ages of twenty and forty. Women appear to die more frequently from it than men; this greater liability to peritonitis in the female arising, perhaps, from the great sympathy between the uterus and the peritoneum —a sympathy which is strongly marked, not only at the period of menstruation, but also at the time of parturition. At the latter period, indeed, puerperal peritonitis often becomes contagious among parturient females.

Diagnosis.—The pain being greatly increased on pressure, and the pulse rapid, together with the general uneasiness and evident danger of the patient, readily distinguish peritonitis from colic. Its salient points of difference from enteritis will be noticed under that head.

Prognosis.—Partial peritonitis often terminates without in any sensible degree impairing the general health. Thus we often find extensive adhesions of the liver without any marked symptoms, as well as limited opacities of the membrane. In every case, however, in which the structure of the peritoneum is thickened or otherwise impaired, the patient may recover, but generally he relapses and dies of dropsy; for the peritoneum, like all other serous tissues, appears to possess little power of restoration after disease. Every attack of acute inflammation is of grave import, and when pus is effused it is very generally fatal; neither will the patient likely recover if the peritonitis is caused by sub-peritoneal tubercles, typhoid or cancerous lesions.

Treatment.—The treatment of acute peritonitis must be active—the activity of the treatment being proportioned to the amount of pain, the rapidity of the pulse, and intensity of the inflammatory fever, which is frequently marked by the peculiar depressing influence of the inflammation on the heart's action. In the milder forms of the disease, when the pain is bearable, and the pulse steady and under 100, twenty leeches over the abdomen, followed by warm fomentations, together with the administration of opium in frequently repeated grain doses, ought to be the basis of treatment. In the severer forms of disease the first indication is to relieve pain. The stomach may be unable to retain food; and vomiting may be present. Under such circumstances, Dr. Anstie has found great benefit from the injection *per rectum* of a pint of strong meat soup slowly thrown up in three successive portions. About three hours' relief of pain was obtained, when a new injec-

tion of soup being given, the same relief to pain followed, and the pulse fell from 124 to 104. Small quantities of broth and wine could then be borne by the stomach, which were administered every two hours (*op. cit.*, p. 117). All action of the bowels should be prevented for several days. Opium should be given in doses of one to two grains, repeated as often as its effects subside—generally every two, three, or four hours. Morphia may be given in doses of a quarter to half a grain, and similarly repeated. Morphia combined with chloroform, as in the formula for chloromorphine or chlorodyne, recommended in vol. i., p. 502, will be found useful in allaying pain.

Leeches applied to the abdomen is the only method of blood-letting likely to be useful, and a poultice afterwards may be required to encourage the bleeding in sthenic cases. It is not proven that calomel, given for the purpose of inducing mercurialism, has any curative tendency in peritonitis. It has been prescribed traditionally; but the experience of Dr. Taylor regarding mercury in pericarditis, as well as of other physicians regarding the influence of mercury generally in the cure of these inflammations, tends to discard it now from our methods of cure in such affections. Fomentations are to be diligently employed, on the same principle as described under typhoid fever, vol. i., p. 423. Acute peritonitis has also been treated by quinine alone, in large and repeated doses, by M. Beau, at the Hôpital de la Charité. The remedy was given in the proportion of twenty to thirty grains of the drug in the twenty-four hours. When the abdomen can bear pressure, a flannel roller should be firmly applied round the body.

The treatment of chronic peritonitis must be directed by the same principles; but we should be content with effecting a present alleviation of symptoms, and without attempting the removal of the mischief which has already occurred; for in patients that have laboured under chronic peritonitis, and survived many years, the peritoneum has still been found opaque, thickened, and silvery, so that in all probability these alterations are permanent, and not attended with danger.

The diet of the patient in the acute forms of peritonitis should be of the mildest and least stimulant kind.

ASCITES.

LATIN EQ., *Ascites;* FRENCH EQ., *Ascite;* GERMAN EQ., *Ascites*—SYN., *Bauchwassersucht;* ITALIAN EQ., *Ascite.*

Definition.—*A collection of serum or inflammatory fluid effused into the cavity of the peritoneum.*

Pathology and Morbid Anatomy.—Cases of ascites are often examined after death, in which no affection of the peritoneum, or of any organ or tissue, can be discovered. More commonly, however, the peritoneum shows evidence of having been either acutely or chronically inflamed, some viscus diseased, such as the liver or the heart, or some tumor pressing on the large vessels, and causing the effusion which constitutes the ascites, in consequence of the hyper-distension of small blood-vessels.

The most frequent concomitant affection with ascites is disease of the heart and large blood-vessels, to which it is believed that at least one-fourth of all the cases of ascites is owing. In these cases the cavities of the heart are often enlarged, and their walls either hypertrophied or atrophied, or the valves are ossified, or their action otherwise impeded, and the aorta may be pouchy—its elasticity and contractility being impaired by calcareous or other degeneration.

Morbid states of the liver and spleen are the next most frequently associated affections. These organs may be found in every possible state and stage of disease.

In general, *anasarca* accompanies *ascites.* It is an infiltration of serous fluid amongst the elements of the general connecting or areolar tissue of the body, passing, therefore, up through and amongst the more loosely connected parts of the body generally, such as between the skin and the muscles. In these cases the areolar tissue is found in various states: in some cases the interspaces or areolæ are greatly enlarged, while the tissue itself, generally thickened, tears most readily in some cases, while in others it is not only greatly thickened, but also greatly indurated. The fluid which it contains is generally limpid and watery, composed merely of the serous part of the blood; while in other instances the fluid is viscid, contains lymph, and the organizable elements characteristic of inflammatory origin.

The quantity of fluid contained in the abdomen in cases of

ascites varies from a few ounces to many gallons: three to four gallons are by no means unusual, and as much as eighteen gallons are said to have been drawn off at one time by the operation of *paracentesis*. The quality of this fluid is very various. In colour it is generally green or yellow; in consistence viscid, often containing so much coagulated matter as to be incapable of flowing through the canula.

Symptoms.—The symptoms of ascites are extremely well marked, but vary in some degree according to the cause, so that it is better to give, first, a general outline of its more prominent features, and afterwards to point out those particular symptoms which indicate the cause from which it springs.

In ascites, if the quantity of fluid effused be considerable, the abdomen is distended and shining, with a number of large superficial veins creeping over its surface. From the weight of the abdomen, the gait of the patient is upright, like that of a pregnant woman; and if anasarca be present, he generally walks with his legs widely apart. In bed he is unable to lie down, on account of the fluid in the abdomen gravitating towards the chest and compressing the lungs, so that he is obliged to be raised towards the head and shoulders. If the anasarca be limited to the lower extremities, the upper portion of the body is in general greatly emaciated, the sharp and pinched features and the withered arms forming a striking contrast to the protuberant abdomen and swollen legs. On the contrary, if the *anasarca* be general, as the true signification of the word implies, the trunk, the arms, the hands, the eyelids, and face generally, are tumid, and swollen to a most unsightly degree. The urine is often defective in quantity, but is sometimes natural and sometimes in excess. The skin is dry, and the patient thirsty; his appetite greatly impaired, and his spirits generally greatly depressed.

The progress of the disease is seldom accompanied by any severe constitutional symptoms; but at length the legs and scrotum become greatly distended, and often inflame, so that the patient sometimes ultimately dies from gangrene of these parts. Again, bronchitis may take place, or the urine may become nearly suppressed, and similar effusions may occur in the cavities of the pleuræ.

The favourable circumstances are, the secretion of urine being re-established and becoming natural, the subsidence of the anasarca and of the ascites, and then a gradual return to health.

The presence of water in the abdomen may be determined by percussion of that cavity; and the best mode is to place one hand on the abdomen, and to give a sharp but gentle tap on the opposite side with the fingers of the other, when, if water be present, a fluctuation will be felt. If, however, the quantity of fluid be small, the fluctuation is best felt by percussing the side of the abdomen from before backwards. The existence of fluid in the areolar tissue of the trunk or extremities is determined by the finger leaving a mark or "pit;" and the fluid being thus displaced, the part does not recover its original form and fulness for some seconds, and is said "*to pit on pressure.*"

The ascites may form suddenly, and the abdomen of the patient may be distended in a few hours, or the fluid may take weeks or months to accumulate. The duration of the disease is indefinite. If the effusion be general, the patient's life may terminate in a few days; but more commonly the affection is chronic, and the patient survives many weeks or months. Such are the more general phenomena of *ascites* and *anasarca;* but it is now necessary to pass to those particular forms which constitute its varieties.

Ascites sometimes results from the large effusion of serum which is poured out occasionally into the cavity after undue exposure to cold and wet. To this form the name of *active ascites* has been given, and although such cases have not the marked symptoms of inflammation, such as pain, yet the febrile re-action which generally accompanies such cases, and the fact that they yield to those remedies which subdue the inflammatory tendency, indicate such a connection. There are cases of a similar kind in which the *ascites* obviously results from *chronic peritonitis;* and now, although the patient sometimes suffers much pain, more commonly this symptom is wanting, or only occurs in occasional paroxysms. In this latter form the patient generally appears to die from the conjoint effects of anasarca and of ascites. The urine is scanty, but for the most part free from albumen in both these forms of disease.

The forms of *ascites* resulting from the following causes are examples of *passive* ascites, because they are all explicable by the fact that the cause tends ultimately to retard the flow of blood through the system of the *vena portæ.*

A *diseased heart,* or diseased state of the aorta, is often the

primary cause of ascites. The heart's sounds, its impulsion, together with the character of the pulse, will indicate the particular lesion under which the patient labours, as explained under cardiac diseases. Dropsy from this cause may first show itself either by effusion into the abdomen, or into the areolar tissue of the lower extremities, causing *anasarca.* When effusion has taken place, it is often remarked that the action of the heart becomes more regular, its impulse more natural, the pulse slower and steadier, while perhaps the murmur also may disappear. This apparent amendment, however, is fallacious: the dropsical symptoms increase, effusion takes place, first into one cavity and then into another, so that the patient seldom long survives this fatal symptom. The urine in this form of dropsy is generally deep in colour, small in quantity, and of a healthy density.

Diseases of the liver offer the best illustration of how morbid states obstructing the *portal* circulation are the main causes of *passive* or *mechanical* ascites. When ascites arises from a *diseased liver,* that viscus is generally enlarged in the left lobe, when it is enlarged; but mere enlargement is not a common condition giving rise to ascites. The liver in most instances is smaller than usual; it is contracted and condensed, so that its shrunk and diminished bulk compresses the portal circulation. The condensation is also of a peculiar kind. It generally results from a compression of the proper hepatic substance, by a contracting tendency in the connecting fibrous tissue which accompanies the portal vessels—namely, the *capsule of Glisson,* and giving rise to the condition known as *cirrhosis,* or *hob-nail* liver. The ascites in this case has no new feature, except that the patient may or may not be jaundiced. In the former case all the fluids effused are of a yellowish or greenish-yellow colour. The urine also is loaded with bile, which is generally turned green by the addition of nitric acid; while in a smaller number of cases the bile appears to be in a peculiar state of combination with the urine, so that the acid has now no effect on it; the urine likewise is always small in quantity, much loaded with the usual salts, and of a high density. The bowels are difficult to act upon, and the patient is liable to severe abdominal pains, simulating chronic peritonitis. The pulse continues throughout the disease for the most part natural, but the patient usually falls into a typhoid state, from which there is no recovery.

In ascites associated with disease of the *spleen*, the viscus is uniformly enlarged, and can readily be felt occupying the left hypochondriac region, and thus the cause, though not its exact nature, may be determined; for we have no diagnostic symptoms denoting whether the spleen be simply hypertrophied or in a cancerous or tuberculated state. The early symptoms are similar to those which occur in dropsy from disease of the liver, and, for the most part, are secondary to hepatic obstruction; and the termination of the disease, if the patient dies, is generally by hæmorrhage from the stomach and bowels, often so profuse as to amount to many pints in a few hours, greatly exhausting the patient, and hastening the fatal issue.

In dropsy from disease of the *kidney* the urine may or may not contain *albumen;* but in the great majority of cases it does so. When albumen is absent (as the chronic forms of diseased kidney are all devoid of pain), we are unable to determine either the seat or the nature of the disease with which ascites is associated, and the ascites is consequently in general attributed to an affection of the peritoneum, of the liver, the spleen, the kidneys, the heart, or other viscus.

Causes.—These have been already sufficiently indicated, and may be summed up as comprehending undue exposure to sudden changes of temperature, loss of blood, obstruction of the portal circulation from morbid states of the liver, or spleen, or heart, and especially from dilatation of the chambers of that organ. The lesions of the kidney associated with ascites generally exercise their pernicious influence through the diseases of the heart which supervene during their course. Such cases prevail most between the ages of twenty and forty-five, while cases from disease of the heart and liver are most common from the ages of forty to sixty.

Diagnosis.—Ascites is readily distinguished in the male from every other intumescence of the abdomen by the fluctuation on percussion. In the female it can only be confounded with pregnancy, or with *ovarian dropsy*, which consists in the accumulation of fluid in one or more cysts, generally multilocular, within the substance of the ovary, or in a serous cyst connected with the uterine appendages.

The grounds of diagnosis may be thus shortly stated:—

1. The uniform and symmetrical appearance, and general in-

creased breadth across the flanks, possessed by the abdomen in *ascites* from the first, contrasted with the general *one-sided* growth of an ovarian tumor.

2. Percussion gives useful diagnostic results when performed in the different positions in which a patient may be placed. In ovarian dropsy dull sounds are fixed and invariable in one place, whatever position is assumed by the patient. In ascites the dull sound follows the gravitating fluid in all positions.

The development of hydatids in the abdominal cavity may also simulate ascites, and so may an over-distended bladder.

Prognosis.—The *prognosis* in anasarca, in young persons not labouring under any organic disease, is always favourable. If, however, it be consecutive to organic disease, a fatal termination is ultimately to be feared.

Ascites arising from indeterminate causes is often recovered from, but no case is free from danger, the peritoneum often being so much impaired that the function of absorption ceases to be effected through it, and so the disease resists the action of all remedies.

Ascites depending on moderate inflammation of the peritoneum is often recovered from, and especially if the inflammation depends on the action of exposure to cold or to paludal poison.

Ascites with albuminous urine, arising from mere functional disorder of the kidney, is generally recovered from; but if the structure of the kidney be impaired, the disease is always grave, and generally fatal. In a few cases, however, the disease subsides, and the patient may continue well for two, three, or four years, when he generally relapses and dies.

Ascites from disordered function of the heart is often recovered from; but if it depends on diseased structure, either of the heart or large vessels, some temporary amendment may take place, while the patient quickly relapses and finally sinks.

Ascites depending on diseased structure of the liver or of the spleen is rarely recovered from unless the primary disease be cured.

Treatment.—When ascites occurs without any obvious organic cause, and without albumen in the urine, the best remedy is the *bitartrate of potash*, administered in divided doses, as one drachm three times a day, or every six hours, or in one large dose, as half an ounce, combined, if the patient's bowels be confined, with ten to fifteen grains of *jalap*. When the smaller doses are used, it is

often exceedingly useful to add ten grains of the *citrate* or *tartrate of iron* to each dose. If these remedies should fail, one-sixth to ~~half~~ a grain of the *extract of elaterium* every night, or every other night, may be given.

There is a form of ascites, without any obvious organic cause, in which there is accompanying anasarca. Under these circumstances *squills* appear to afford most relief; and by giving five to eight grains of the *pulvis scillæ* three times a day, the dropsy is generally relieved, and the patient sometimes cured. If the stomach be irritable, *half a grain of opium* should be added to each dose so that the remedy may be retained.

Should the ascites arise from simple inflammation of the peritoneum, this form of dropsy in general yields to leeches and fomentations to the abdomen, together with the administration of a mild saline aperient every six hours.

When the ascites arises from disease of the heart, the kidney being sound, and the urine free from albumen, the treatment must have reference to the nature of that disease. If the valves of the heart are diseased, the patient, though he cannot recover, may be greatly relieved by the administration of tonics, stimulants, and saline or drastic purgatives. An *ounce and a half of camphor mixture, with a drachm* of the *spirit of nitrous æther, fifteen minims* of the *tincture of hyoscyamus*, and *a drachm* of the *sulphate of magnesia*, will form a draught which, taken *three times* a day, will often greatly reduce the dropsy. When the stomach will bear it, the *tincture of squills* (ℳx. to ℳxx.), with a *drachm* of the *acetate of potash*, has occasionally succeeded. Small doses of *elaterium*, as one-eighth to one-fourth of a grain three times a day, is a medicine that is also sometimes useful.

The ascites may be caused by disease of the liver; and should that organ be merely inflamed or hypertrophied, without other alterations of structure, the dropsy may disappear with the cure of the hepatic disease. The treatment is by bleeding, if the hepatic lesion be the congestion of inflammation, and the neutral salts, as the sulphates of magnesia or of soda; or should they fail, by moderate doses of calomel. When, however, its structure is otherwise altered, the patient is seldom cured; but the disease may still be alleviated and life prolonged. In this form of ascites the patient suffers greatly from abdominal pains, which are relieved most effectually by fomentations. In these cases, also,

the bowels are often greatly constipated, and require the most powerful drastic purgatives, as the black draught, castor or croton oil, or elaterium. In this form of dropsy, however, the peritoneum becomes more impaired in its power to absorb the fluid than in most of the others; the fluid is therefore seldom reduced, and the patient generally requires the last imperfect resource of art—namely, tapping, or *paracentesis.*

Ascites depending on enlarged spleen is also difficult of cure. If the spleen be simply hypertrophied, the *bromide of potash* and the *iodide of potassium,* in doses of five to eight grains three times a day, have been recommended. The patient, however, often dies from hæmorrhage from the stomach after all the more prominent symptoms have been relieved. My friend and colleague, Dr. Maclean, informs me that rubbing the *biniodide* of mercury, in the form of an ointment, on the skin over the region of the enlarged spleen, has a very marked beneficial effect in reducing the enlargement. The ointment is to be rubbed into the skin while the patient sits before a strong fire, or in the rays of an Indian sun; and, as a further evidence of efficacy, the invalid soldiers invariably ask to be furnished with a supply of the remedy when they go from the hospital. The *biniodide of mercury,* similarly used, has been noticed as of great service in reducing the swelling of a goitre (vol. i., p. 859).

The dropsy which often occurs in young chlorotic women, in whom the urine *contains albumen,* the kidney being healthy in structure though disordered in function, is generally curable,—the most efficient remedy being the *bitartrate of potash* in *drachm* doses three times a day. It acts as a diuretic and as a purgative in these cases.

Section II.—Diseases of the Hollow Viscera of the Abdomen.

STOMACH DISEASES.

Definition.—*Under this indefinite heading it is proposed to notice some morbid states of the stomach, expressed by functional disorders, lesions of texture, or both combined.*

Pathology and Morbid Anatomy.—The organic diseases and functional disorders of the stomach have been recently elucidated in

this country by the researches especially of Drs. Budd, Handfield Jones, Chambers, Leared, and Brinton. Nevertheless, the morbid changes which are known to occur in it are still but very imperfectly connected with the expression of clinical phenomena. "There is no part of the body," says Dr. Chambers, "of which we hear so much from our patients, and are able to communicate so little knowledge in return, as about the stomach; and truly," he also observes, "it is an ill-used viscus—flattered in metaphor and insulted in fact." It has been regarded as a mere bag, a mere mill, or a mere chemical laboratory for the solution of substances; yet still all such similes and metaphors, as Hunter remarked, explain nothing; and he wisely insisted that it is a viscus *sui generis*, with definite functions to perform; in short, a "stomach is a stomach." Its morbid states are similarly peculiar to itself and to its functions.

Instead of describing a series of disorders one by one, and by names such as *gastritis, dyspepsia, hæmatemesis,* and the like, it may perhaps be more instructive to write a condensed statement of that positive knowledge so satisfactorily established by the prolonged and well-directed labours of those men just mentioned. Such a method will better furnish those grounds for diagnosis by which clinical phenomena, often of the most painful, prolonged, and harassing kind, may be connected with some understood morbid state, and so furnish some fixed and definite principles by which the treatment of stomach diseases may be directed.

The morbid states of the stomach, ascertained after death to have existed during life, and in some measure to have been expressed by certain symptoms, may be enumerated as follows:—(1.) *Softening of tissue;* (2.) *Glandular degeneration of the proper mucous substance;* (3.) *Congestion;* (4.) *Forms of inflammation tending to exudations and destruction of parts, or condensations of tissue, especially about the pyloric orifice;* (5.) *Ulceration;* (6.) *Carcinoma.*

Abnormal functional states are expressed by—(1.) *Vomiting, associated with lesions of other organs.* (2.) *Deficient secretion of gastric juice.* (3.) *Fermentive processes (alcoholic, butyric, or lactic, tending to the development of entophytes, such as sarcinæ.* (4.) *Indigestion, associated with and depending upon*—(a.) *Morbid states of those viscera which are conjoined with the stomach in the processes of digestion, such as the liver, pancreas, and small intes-*

tines; (b.) *Imperfect action of the kidneys, as in Bright's disease;* (c.) *Defective or diminished morphological changes during the processes of nutrition in the tissues, generally expressed by altered secretions and excretions, as in many constitutional diseases;* (d.) *Indigestion, associated with pyrosis and increased secretion of the juices of the stomach and salivary glands, and with cutaneous disorders, such as urticaria;* (e.) *Indigestion, associated with drunken habits.* The diseases of the stomach with which one or more of these organic or functional states may be associated are,—*gastritis, chronic ulcer, hæmatemesis, perforation, dilatation, stricture, gastric fistula, hernia, cancer, colloid, tumors* (non-malignant), *sarcinæ, injuries, laceration, dyspepsia, gastrodynia, pyrosis.*

The essential juices of the stomach and of the intestines play a most prominent part in determining the nature of these diseases; and some of the most important principles of treatment are based upon the physiological relations of those juices, and upon the fact that while the process of digestion of food is only commenced in the stomach, it is completed in the alimentary canal by the influence of the intestinal fluids. There is a *stomachal digestion,* in which the gastric juices, with the saliva and mucus of the stomach, play the most prominent part; and there is an *intestinal digestion,* in which the intestinal juices, composed of the *biliary, pancreatic,* and *intestinal secreted fluid,* play the most prominent part. It is also an important fact, especially insisted upon by Dr. Chambers, having been proved by the experiments of Bidder, Schmidt, and Handfield Jones, that the intestinal digestion may be made to do more or less of the work of stomachal digestion, so that the exercise of the function of the stomach may be spared when necessary, and the food encouraged to pass from it into the bowels, to be digested entirely by the intestinal juices.

The reader is referred for information regarding the properties and constituents of the gastric juice to the text-books on physiology; but it is necessary here to advert to the fact, recently established by the experiments of Schmidt and Bidder upon animals, and the observations of Grünewaldt upon an Esthonian peasant who suffered from a stomach fistula, that there is a constant circulation of an immense quantity of fluid through the mucous membrane of the alimentary canal, necessary to the solution and absorption of food from its interior. It is now

also well ascertained that from the gastric glands the principal part of the solid animal matter of the gastric juice is derived, and which is the exciting cause of the solution of albuminoid substances in the stomach. The solid matter is regarded as a *gastric ferment*, and has been variously named as such, and also as *rennet*. But water constitutes the chief bulk of the juice; and from the experiments and observations alluded to, it is known to perform a most important function. It is continually poured forth from the surface of the mucous membrane in vast quantities, to the extent of between *a fifth* or *a quarter* of the *weight* of the *body*, and as constantly returns to mix again with the sanguineous fluid. This secretion of fluid is constantly going on from the internal alimentary mucous surface. It mixes with the dissolving food in the alimentary canal, and takes up those particles of it which it is calculated to hold in solution, loaded with which it returns again to the sanguiferous, chyliferous, and lymphatic circulations. Thus a "poor" watery fluid is constantly being sent forth, to return "*laden with wealth*," and so to minister to the nutrition of the body. This has been described as an internal mucous circulation of fluid within the body; and the arrest of this interchange, with the great retention of water, has been shown by Virchow and Parkes to be a constant condition in the febrile state (see vol. i., p. 58).

I. **Softening of the Stomach.**—It was first announced by Hunter, and subsequently confirmed by the experiments of Spallanzani, Wilson Philip, and Carswell, that the stomach under certain conditions as to temperature and properties of the gastric juice, especially at death, and immediately after that event, may be dissolved or digested by the secretions poured forth from its own secreting glands. In certain diseases, also, of a catarrhal kind, it has since been ascertained that potent gastric juice is sometimes secreted by the empty stomach, or that lactic acid being freely generated from the saccharine principles of food, forms with the mucous membrane an efficient digesting mixture; and not unfrequently a softening of the stomach may be predicted with tolerable certainty by a peculiar train of symptoms, and resulting from the presence of free gastric juice, or of a digesting acid, in the otherwise healthy stomach (BUDD). Dr. John Gairdner, of Edinburgh, however, so long ago as 1824, inculcated a similar doctrine. He observed, in a numerous series of cases of children,

that a peculiar action of the gastric and intestinal mucous membrane, analogous to inflammation, weakened the texture of the organ, and rendered it morbidly susceptible to the action of the gastric juice during life (*Edin. Med.-Chir. Trans.*, vol. i., p. 311). It was in opposition to this view that Carswell performed his experiments, an account of which he published in 1838 ; so that the testimony they undoubtedly afford regarding *post-mortem softening*, as the only kind of softening, must be received with qualifications. The observations of Dr. John Gairdner and Dr. Budd evidently show that the probability of softening may be predicted during life, although Dr. Budd is of opinion that the softening does not take place till after death. A similar opinion is expressed by Andral. The diseases in which it is so apt to occur are those in which there is much cerebral disturbance and increasing debility and emaciation before death, and from such diseases as *typhoid fever, cancer of the uterus, or peritonitis,*—in infants who die of *tubercular hydrocephalus;* in deaths from exhaustion from inflammatory diseases of the brain, when vomiting is a constant symptom ; in persons who die from phthisis and from ulcer of the stomach. The symptoms from which the softening may be predicted are, that when, along with any of these diseases, there is much disorder of the stomach, such as pain and tenderness at the epigastrium, loss of appetite, thirst, frequent vomiting of acid fluids, and nausea, the lesion may be expected to be found after death. In the common form, as Hunter described it, the mucous membrane towards the splenic end is thin, and for the most part stained by hæmatine, very slippery, and appearing as a dark film gliding over the submucous tissue. The tubes appear under the microscope to be a good deal altered, chiefly by solution of their epithelium, while dark grains of melanic matter are deposited between the tubes. The amount of probable knowledge we possess respecting softening of the stomach is thus expressed by Dr. Handfield Jones :—

1. There are two forms of softening: one, the commonest by far, which is simply the result of the action of the acid contents of the stomach upon its own dead tissue ; the other, the consequence of a peculiar change taking place in its glandular structure, which generates a powerful acid, dissolving, corroding, or destroying the surrounding tissue.

2. The latter form may occur either with an empty or a full

state of the blood-vessels of the stomach, the softening part of which will accordingly be either quite pale or of a dark blackish tint.

3. This same form occurs in a great variety of morbid states, which seem to have only this in common,—that they are attended with great depression of the vital powers.

4. It is more common in children than in adults, on account [probably] of the greater delicacy and less resisting power of the system.

II. **Glandular Degeneration of the Proper Mucous Substance** is by far the most common of the organic lesions—existing in 72 out of 100 cases examined by Dr. Jones. It is expressed in a variety of forms, the most marked of which are—(1.) Peculiar morbid changes in the tubes, probably analogous to the process which occurs in the tubes of the kidney in Bright's disease, where the molecular contents of enlarged epithelial cells increase, ultimately leading to complete destruction, so that the tubes become filled with the debris of this destruction; (2.) Melanic and fatty deposits in the epithelium; (3.) Interstitial deposit of nuclear and fibroid exudation at the expense of the gland-substance, so that hypertrophy of tissue may exist, along with—(4.) Atrophy of the tubular epithelium of the tubes themselves, and of the solitary glands—a form of cirrhosis generally associated with alcoholism, and described by Dr. Wilson Fox.

There is thus, on the one hand, in some instances, an utter destruction of the tubular glands—actual loss of substance without replacement; on the other hand, there is no actual loss of substance, but a replacement of elements by the deposition of granular matter within the tubes, without diminution or alteration of their form. That these partial degenerations, even when they extend over a considerable portion of the stomach, do not materially interfere with the ultimate digestion of food seems to be established by the cases described by Dr. Jones, deficiency of stomachal digestion being compensated for by increased vigour of intestinal digestion. Such loss of the gastric glands appears thus to have little influence over the vital acts, so that it is rare to find any evidence of the existence of such lesion during life, even when the degeneration is very extensive. I believe, however, from observations upon the intestinal mucous membrane in analogous glandular degeneration, that in cases characteristic of intestinal

wasting, associated with anæmic states, as described at p. 80 of this volume, the mucous membrane throughout the whole alimentary tract is similarly affected, and that an examination of the mucous membrane of the lips, and buccal membrane of the mouth, indicates with great probability the change below, just as the condition of the tongue is the index from which we judge generally of the functional state of the alimentary canal. The general symptoms of such cases are undoubtedly anæmia and debility, without any obvious organic cause, often associated with vomiting or nausea in the morning, no desire for food, and a sensation as if it never was effectually swallowed, but stuck at the diaphragmatic entrance of the stomach, causing the peculiar feeling of weight which attends indigestion, and the abundant generation of gaseous fluids. It has been also well remarked by Dr. Chambers, that such symptoms indicate a participation of the whole alimentary tract in such lesions, and that where the lesion is merely confined to the stomach, the absence of such symptoms are sufficiently accounted for by the compensation to stomachal digestion afforded by the healthy intestinal tract.

III. **Forms of Congestion.**—These may be described as *passive congestions;* or as *active congestions* associated with inflammatory lesions.

The *passive* form of *congestion*, like the passive form of *ascites*, is explained by the physiological phenomenon now recognized as perfectly familiar—namely, that the freedom of the transit of blood through any part is in a great measure dependent upon the healthy performance of its functions, and of the function of those parts immediately associated with it—so that, if function is arrested, the circulating current is arrested also, and the blood reverts, or is thrown back, to those intervening parts in the course of the circulation between the site of obstruction and the force propelling the blood. Thus blood ceasing to flow freely through the lungs reverts, or is thrown back, upon the right side of the heart; blood ceasing to flow freely through the liver is thrown back upon the stomach, upon the one hand, or upon the spleen by the gastro-epiploic vessels, expressed by congestions of these viscera; or, on the other hand, the portal system being obstructed, the blood reverts to the membranes of the intestines, and expresses such an occurrence by ascites, as well as by gastric venous congestion. Any mere mechanical impediment, therefore,

which prevents the blood returning from the stomach towards the heart will induce congestion of the stomach.

The immediate effects of such congestion upon the functions of the stomach are sufficiently expressed by symptoms such as *hæmatemesis*, or the vomiting of blood; the secretion of the gastric juice also is diminished, the stomach can digest less food, and requires longer intervals of rest between the meals.

The persistence of such passive forms of congestion further leads to the deposition of melanic matter, and to disintegration of the tubular glands of the stomach, the formation of ulcers in its mucous membrane, just as varicose ulcers are established in the limbs from the passive congestion which attends varicose veins in the lower extremities, by impairing the nutrition of the part, as explained by Simon, Budd, and Jones.

Besides congestion from mere mechanical impediments, there are congestions of the stomach which have been described as *vicarious*—that is, as taking the place of congestions which ought to occur in other parts—such, for example, as when it occurs along with hæmorrhage in women from stoppage of the catamenia; and which has been observed to occur in yellow fever, in malignant cholera, and in typhoid fevers. Congestion also arises during the development of—

IV. **Forms of Inflammation tending to Exudations and Destruction of Parts, or Condensations of Tissue, especially about the Pyloric Opening.**—*Idiopathic gastritis* is an exceedingly rare disease. When inflammation of the stomach does occur, it is generally the consequence of direct injury from irritant or corrosive poisons. Dr. Jones has never met with a case of acute idiopathic gastritis. Louis examined five hundred bodies without finding a single instance. Andral, however, relates several cases in his *Clinique Medicale;* and it is stated by Dr. Robert Williams, that during the whole of the Peninsular war not more than six cases were reported among the troops, although exposed to every species of privation, and addicted as they were to its most efficient exciting cause—the use of spirits in every form in which alcohol could be got to drink.

The difficulty of exciting acute inflammation in the stomach is well shown by experiments upon animals, and by the often long escape of the polyphagist, who swallows knives and watches, and all sorts of heterogeneous things; and of the Indian who passed

many times daily a blunt sword into his stomach with impunity, till at last its coats were pierced, and he died. The stomach, also, we find will bear tea or coffee of an almost boiling temperature, followed perhaps shortly afterwards by a quantity of ice. One of the persons resident at the Eddystone Lighthouse at the time it was burnt, in 1755, swallowed a quantity of molten lead, which accidentally dropped into his mouth when looking from below upwards, to observe the progress of the fire. But even after this intensely hot substance had passed into his stomach, he lived several days, having been taken to the Plymouth Hospital, where he was attended by Dr. Sprey, who describes the case in the *Transactions* of the Royal Society. His attendants hardly believed his story possible; but on examining him after death, a lump of lead, weighing 7 oz. 5 drs. 8 grs., was taken from the stomach.

The simplest and most frequent form of inflammation of the stomach is that which is brought about by excess in eating or drinking, especially of alcoholic drinks. It is characterized by active congestion and an excessive secretion of mucus in the stomach—a condition which Dr. Jones describes under the name of *gastric catarrh.* Dr. H. Jones thus describes its phenomena:—

"It occurs under the same influences as catarrh of the air-passages or conjunctivitis, and often either co-exists with these affections or succeeds them. Its anatomical characters are, distension of the capillaries, and abundant secretion of an extremely tenacious, clear, whitish bile or blood-tinged mucus. At the commencement of the disease, or during exacerbations, the hyperæmia is a marked feature; but the presence of abnormal mucus is a more constantly observed and certain character. Congestion of a passive kind powerfully predisposes to this disease, and is commonly conjoined with it. Watery, slightly mucous, feebly acid, or neutral fluids are secreted, and often ejected, constituting pyrosis. It does not tend to the destruction of the glandular tubes" (*Pathological and Clinical Observations respecting the Stomach*, p. 70).

There is a comparatively rare form of inflammation, described by Dr. Budd, in which coagulable lymph is effused into the submucous areolar coat, and, hardening and contracting, forms a dense gristly mass, binding the mucous membrane to the parts beneath. Round the pyloric orifice such a morbid state often acts as a permanent stricture. It is a condition almost invariably

the result of spirit drinking, and seldom occurs before the age of forty.

V. **Ulceration.**—The observations of Drs. Chambers, Gairdner, Habershon, Jones, and Brinton, have shown this lesion to be by no means uncommon. *Simple, chronic,* and *perforating* ulcers are described. The ulcer is generally solitary and deep, seldom larger than a shilling, circular or oval in shape, its edges clean, sharp, and well defined, as if punched out. It is generally situated along or near the lesser curvature, and usually nearer the pyloric than the cardiac orifice, and more frequently on the posterior than the anterior wall. It is more common in women than in men, nearly in the proportion of two to one. It is especially a disease of middle and advanced life, although Dr. Budd once met with it in a girl of fourteen. Dr. Brinton records two cases under the age of ten years. It is relatively more frequent amongst the poor than amongst the rich; and is especially found in maid-servants between the ages of eighteen and twenty-five.

It tends to prove fatal by—(1.) *Perforation;* (2.) *Hæmorrhage;* (3.) *Exhaustion.*

When the ulcer eats its way through, the aperture gradually narrows, as it reaches the peritoneum, to little more than a point, which corresponds to the centre of the ulcer. The peritoneum inflames at this spot, and sloughs, when perforation is completed by the separation of the slough. An important sanitary effort is here made by the adhesion of the peritoneum surrounding the slough to some opposed surface, such as to the liver, pancreas, colon, or abdominal wall. Fistulous openings may then be established, so that food escapes from the stomach when such passages communicate with the alimentary canal. A most comprehensive record of such cases is published in *The Edinburgh Monthly Journal* for July and August, 1857, by Dr. Charles Murchison, physician to the London Fever Hospital and to the Middlesex. According to Dr. Brinton's observations, about 13·4 *per cent.* of cases of ulceration terminate by perforation; and he considers that the liability to perforation decreases as life advances; and, as Dr. Crisp first showed, during the ages from fourteen to twenty it is most frequent. The patient may survive the first shock of the accident, ultimately to succumb to the combined effects of peritonitis and gastric exhaustion.

Hæmorrhage, while it is one of the most frequent and important symptoms of ulcer, is also a mode of fatal termination. It generally occurs soon after a full meal; and about from $3\frac{1}{2}$ to 5 *per cent.* of the cases prove fatal in this way.

"This long and exhausting malady," says Dr. Brinton, "predisposes the constitution to a variety of other diseases, and renders unusually fatal many of those attacks of illness which, in the course of years, very few persons altogether escape."

Symptoms of Gastric Ulcer.—"The malady is announced by disturbances of gastric digestion: at first by mere uneasiness and pain; then nausea and vomiting, or regurgitation, that expel the food previously taken; or a tasteless or acid watery secretion. At this stage of the disease it is sometimes cut short by the occurrence of perforation, with its sequel of fatal peritonitis. Failing such an accident, the dyspeptic symptoms are next complicated by hæmorrhage from the stomach; sometimes a sudden and dangerous gush, oftener a slow and intermittent drain of blood. The anæmia produced by this hæmorrhage is generally associated with a cachexia which seems to be essentially independent of it; being chiefly the result of the inanition necessarily implied by frequent vomiting of the food, or by large destruction of the gastric mucous membrane, and consequent impairment of its function. In young females another symptom is often present, in the form of more or less complete amenorrhœa, which may be associated with either of these two states of anæmia or cachexia; in other words, may be connected with ulceration, with hæmorrhage, or with both. The gradual acquisition of all these symptoms conducts the disease, in a variable period, to a climax, whence we may next briefly trace it towards its termination. Retaining the liabilities to death by perforation, by hæmorrhage, by vomiting, and by exhaustion, which the above organic results of ulceration severally imply, the lesion often ends by one of these modes of dying, or by two or more of them in combination. In other cases a spontaneous subsidence of these symptoms, in something like the inverse order of their occurrence, announces a recovery; or a similar amendment is only effected by a careful medical treatment, such as quite entitles us to dignify it by the name of a cure" (*Med.-Chir. Review*, p. 159, July, 1856).

The character of the pain is peculiar, at first being little more than a feeling of weight or tightness, of a dull character, and continuous. It gradually becomes intensified into a burning or gnawing sensation, which produces a kind of sickening depression. It generally comes on from two to ten minutes after the ingestion

of food, and remains during one or two hours, which correspond to the period of gastric digestion, after which it gradually subsides, or, if vomiting empties the stomach, it also invariably ceases. The pain is generally expressed at the centre of the epigastrium, or at the middle line of the belly, immediately below the extremity of the ensiform cartilage, often confined to a mere spot, and rarely to a space more than two inches in diameter. A dorsal pain, first described by Cruveilhier, is also subsequently established, generally in a few weeks or months after the epigastric pain. It is expressed by a gnawing sensation, interscapular, or from the spine of the eighth or ninth dorsal to that of the first or second lumbar vertebra. Pressure in the epigastric region is sometimes unbearable, and, for obvious reasons, must be applied with the utmost care and delicacy.

Vomiting usually occurs when the pain reaches its height; and, completely emptying the stomach, generally affords relief. For further details of symptoms, the reader is referred to the admirable papers of Dr. Brinton, from whom the statements have been condensed.

VI. **Carcinoma.**—The cachectic state which ulcer of the stomach ultimately induces has caused many cases of simple ulcer of the stomach to be recorded as cancer; so that it has been a popular belief that cancer of the stomach is a very common disease, till the inquisitive researches of Dr. Brinton have shown that the mortality attributed to gastric cancer is by far too great. Of necropsies performed at four of the great London hospitals, only about 1 *per cent.* of the total mortality is due to this lesion. The disease is thus far less frequent than ulcer of the stomach. Next to the female *breast*, the *stomach* seems, in this country, to be the part most liable to cancer, and the male is more frequently affected than the female—"a fact," writes Dr. Brinton, "which it is impossible to avoid connecting with the exclusive amenability of the female to the *mammary* and *uterine* localization of cancerous disease."

The orifices of the stomach are the parts which are almost exclusively the seat of cancerous lesions.

VII. **Abnormal Functional States.**—These are due—*First*, to what have been called sympathetic relations with other organs, themselves in a morbid state, and which are now explained by the phenomena of "*reflex action*." Examples of this may be

referred to in the *vomiting* which attends irritation of the *lung, brain, liver,* or *uterus. Second,* a scanty secretion of the gastric juice, characterized by slowness of digestion and long retention of food by the stomach; prolonged distress after eating, especially of weight and uneasiness at the pit of the stomach; peculiar tendency to decomposition of food in the alimentary canal; the evolution of fetid gases; and the appearance of unaltered ingesta in the stools. Often an inheritance by birth, the conditions which tend to produce this morbid state are, mental over-exertion, prolonged anxiety, especially after meals, gluttony, drunkenness, and sedentary habits, and the consumption of more food than the system requires. The "*causes, symptoms, and treatment of imperfect digestion,*" from these points of view, are admirably treated of by Dr. Leared in his little book on this subject.

Urine in Dyspepsia.—Alterations in the free acidity are the most important signs. But in some dyspeptic cases, especially those attended with torpid digestion or with acid vomiting, it has seemed to Dr. Parkes that the urine is more alkaline than usual; so that there may be abnormal acidity as well as abnormal alkalinity in cases of dyspepsia. When the urine is more acid than usual after food, it is also often scanty, and deposits urates and oxalates of lime—conditions associated chiefly with cardialgia, nausea, and frontal headaches. Chloride of sodium is small in amount when digestion is imperfect; and the more that is in the urine, the more perfectly has digestion been carried on. Oxalate of lime-crystals are common in dyspepsia (PARKES, l. c., p. 333).

Treatment.—Congestion, catarrh, ulcer, cancer, and functional states associated with what are called dyspeptic symptoms, or "imperfect digestion," are the causes of stomach diseases for which the physician is called most frequently to prescribe.

When there is reason to believe *congestion* exists, a sparing and easily digested diet is to be prescribed, and total abstinence from fermented drinks is imperatively demanded; and in cases where there is reason to believe that catarrhal inflammation prevails, the blandest food must be given in very small quantities. In severe cases leeches are to be applied over the region of the stomach, and the patient may sip iced water, or suck small pieces of ice, to relieve the thirst.

For obvious reasons ulcers are most difficult to heal, and such cases are to be treated by rest to the stomach, and by dietetic

rather than by medicinal means. The patient must eat in small quantities, and especially of milk compounded with arrow-root, macaroni, semolina, sago, tapioca, biscuit powder, Indian meal, or oatmeal gruel. Iced milk, combined with one-quarter to one-third of lime-water, is particularly recommended by Dr. Chambers; two or three table-spoonfuls of which are to be taken at short intervals, so that about two quarts of milk may be thus used during the day, rendering regular and more bulky meals unnecessary. The lime-water tends to prevent coagulation of the milk, and the milk thereby more readily passes unaltered into the intestines, to be digested by them rather than by the stomach. Dr. Balthazar N. Foster, of Birmingham, has well laid down the details of managing such cases, in "A Lecture on the Treatment of Gastric Ulcer" (*Brit. Med. Journal*, June 3, 1865). The most complete rest possible ought to be given to the affected viscus, by stopping the supply of all nutriment by the mouth, and supporting the patient for several days by nutritive enemata. Perfect quiet in the recumbent posture must be observed, the lips and tongue being moistened from time to time by a little water. The patient may thus be kept for eight or nine days, or even longer, on nutrient enemata alone, when the pain, the irritability of stomach and of the system will cease. The substances Dr. Foster has found most useful for enemata are milk, strong unsalted beef-tea, raw eggs beaten up in milk, occasionally a little brandy, and generally in two enemata daily ten or twenty minims of tincture of opium. The enemata should be as small as possible, from two to six ounces only at a time. The interval to the stomach thus gained of complete rest is of the greatest importance towards success in the treatment of gastric ulcer, and, combined with the restricted diet described, will usually effect a cure in three weeks.

Next to dieting, blood-letting by two or three leeches applied to the region of the stomach about twice a week affords obvious benefit, the patient often gaining weight during their use. Blistering applied to the spine is also said to relieve the dorsal pain. Small lumps of ice may be swallowed if the stomach is irritable, and astringent remedies, especially the *salts of metals*, such as a combination of *iron* and *alum*, are of great benefit; and, as a change, gallic acid, nitric acid, and bitter barks may be administered. Constipation is to be counteracted by enemata. When hæmorrhage occurs, Dr. Budd recommends small bits of ice

to be swallowed, that rest be maintained in the horizontal posture, and that astringent medicines be administered, such as *oil of turpentine, acetate of lead and opium, alum and tannic acid. Oil of turpentine* should be given in doses varying from ten to twenty minims in *cold* water, and repeated more or less frequently according to the urgency of the symptoms. When hæmorrhage is copious, a mixture containing ten minims of *diluted sulphuric acid* and five grains of *gallic acid* in water, taken every two or three hours, is the best remedy (Dr. B. Foster). Dr. Leared justly puts great stress upon the necessity of strict attention to diet in all cases of impaired digestion. If fulness and uneasy sensations are experienced after dinner, less food should be taken at that meal, and more at breakfast, the principle being to apportion the amount of food necessary to sustain the body more evenly over the waking hours than is commonly done. The great fault in the dietetic system of this country consists in the fact that most people are supported mainly by dinner. This meal is consequently too large. The quantity taken at dinner should therefore be resolutely diminished till breakfast is appreciated (Leared, l. c., p. 150).

Special symptoms, common to various morbid states, require special modes of treatment. Excess of acid is best neutralized by *lime-water* or *magnesia*, and the gastric fermentation which is apt to be established may be checked by *brandy* and *various aromatic spirits. Bicarbonate of potash* and *nitrate of potash*, in the proportion of *eight* parts of the former to *one* part of the latter, is useful in cases of habitual acidity; and all these antacid remedies should be taken about three or four hours after a meal. Pills containing from a quarter to half a minim of *creosote*, given with each meal, will in general counteract fetid eructations. It checks that fermentation in which acetic and carbonic acids are formed; while *conium* and *belladonna* are the medicines which better than *opium* allay general nervous irritability.

In cases of slow digestion, with deficient secretion of the gastric juice, the rules of treatment are—(1.) To let albuminoid food be as liquid as possible; (2.) To let the day's allowance be taken in small quantities at regular intervals; (3.) That by the administration of alkalies the food may pass to the intestines, and be digested there, rather than by the stomach. This latter mode of treatment by alkalies, recommended by Chambers, is contrary to

that recommended by Dr. Budd. Both are consistent with physiological facts, and the course to be followed must be determined by the nature of the case. The amount of food taken is of great importance to be attended to in cases of slow digestion. If a fair amount of exercise be taken, the following dietary, slightly modified from that proposed by Dr. Leared, will be found appropriate in such cases:—

BREAKFAST, (8 A.M.)

Bread (stale),.....4 oz.	Mutton Chop, or other meat (cooked) free from fat and skin,...3 oz.	Tea, or warm milk and water and sugar, or other beverage,.....¾ pint.

LUNCHEON, (1 P.M.)

Bread (stale),.....2 oz.	No solids, such as Meat or Cheese.	Liquid,.....¼ pint.

DINNER, (5 or 6 P.M.)

Bread (stale),.....3 oz. Potatoes and other Vegetables,.....4 oz.	Meat (cooked) free from fat and skin,.....4 oz.	Liquid, not more than ½ pint.

TEA or SUPPER (not sooner than three hours after Dinner).

Bread (stale),.....2 oz.	No solids, such as Meat or Cheese.	Tea, or weak brandy and water, or sherry and water, or toast and water, to the extent of ½ pint.

For excellent advice on this subject, the reader is referred to Dr. Leared's book, p. 160, *et seq.;* and to Dr. T. K. Chambers's *Lectures chiefly Clinical.*

Forms of indigestion marked by excessive acidity and heartburn may be relieved by bicarbonate of soda, in doses of fifteen grains, combined with a few grains of nitre, and taken two or three times a day. At the same time, free excretions from the liver and bowels must be sustained by occasional small doses of *blue* pill or *podophyllin,* combined with extract of *colocynth* and of *henbane;* while exercise and diet are duly attended to.

Weakened digestion from over-fatigue may be often restored under the use of *carbonate of ammonia,* conjoined with *compound tincture of gentian,* or with *extract of gentian* in the form of a pill. Extracts of *nux vomica* or *strychnia* are valuable remedies. *Half a grain of extract of nux vomica, half a grain of sulphate of iron, and four grains of compound colocynth pill,* form a combination which, taken early in the morning, generally induces gentle action of the bowels (LEARED). *Compound*

rhubarb pill may be substituted in place of the *compound colocynth pill.*

Indigestion from habitual drunkenness is best relieved by bitter infusions, such as *gentian, quassia,* and *calumba,* singly, or combined in a mixture, so that a dose may be taken two or three times a day, an hour before each meal. Small doses of *opium* or of *morphia* in an ammonia mixture may also be given at bed-time, so as to secure sleep at night.

In prescribing the mineral acids, the following general rule, stated by Dr. Bence Jones, ought to be kept in mind—namely, that the influence of *sulphuric acid* is astringent, while that of *hydrochloric acid* promotes digestion, and of *nitric acid* secretion.

DISEASES OF THE INTESTINES.

Definition.—*Under this heading, as with stomach diseases, it is proposed to notice the more certainly ascertained or common morbid states of the lesser intestinal canal, expressed by functional disorders, lesions of texture, or both.*

Pathology.—The diseases usually named are *enteritis, typhlitis, dysentery, ulceration, perforation, abscess in the sub-peritoneal tissue, fæcal abscess, fistula (fæcal), hæmorrhage, melæna, dilatation, tympanitis, obstruction, stricture, intus-susception, internal strangulation, hernia.*

Although *enteritis* is generally described in text-books, as a rule it is a rare disease, and seldom affects the intestine throughout its whole extent. The several parts under which it is anatomically described are variously influenced by local inflammatory processes. The peculiarities of anatomical organization, such as the various forms of minute glandular parts, determine in some measure the forms by which these organic lesions are expressed. With some modifications, explained by such peculiarities of structure, the *organic* lesions of the lesser intestinal tract are in many respects precisely similar to those described in the stomach; while the functional disorders are indicated by the various forms in which the intestines express irritation or perverted action, such as by *spasms, colic, flatulence, indigestion, constipation,* and various forms of *fluxes* or *diarrhœa.*

I. **Softening of Tissue** has been described in the small intestines similar to that affecting the stomach, but it occurs much less

frequently, and is not expressed by any recognizable clinical phenomena.

II. **Glandular Lesions and Degenerations** are a much more frequent occurrence, if not a constant one, over limited portions of the intestine. The lesions are peculiar, from the structure of the parts; and the *degenerations*, as they are termed, are analogous to those described by Dr. Jones as occurring in the stomach (see p. 833). As in the stomach, so in the intestines there is to be observed in some cases, on the one hand, an entire destruction of the gland-tissue, with actual loss of substance, so that when the mucous membrane is delicately dissected from the muscular parts, it may be viewed as a transparent object with a lens; on the other hand, there may be no actual loss of bulk or of substance, while the structure is nevertheless greatly altered —(1.) By alterations of the contents of the mucous tubes, without change of form; (2.) By an interstitial deposit of fibrinous matter encroaching upon and ultimately obliterating the proper glandular tissue. Thus, externally and in bulk, the parts may seem to be unchanged; but they are found to be materially altered in specific weight, and the range of alteration is considerable. Thus, through lesions or degenerations of the gland-tissue, *atrophy* of the mucous membrane of the intestines becomes expressed in two ways (as originally expressed by Dr. Bucknill with reference to the nerve-substance of the brain), namely,—(1.) *Positive atrophy*, in which the tissue of the gut wastes, while the glandular texture is altered; (2.) The tissue of the gut may not have wasted; on the contrary, it may have gained in bulk, but the glandular tissue has been changed, or been altogether replaced; while (3.) The two conditions may be co-existent.

The observations which appear to me to prove these statements are, alterations in the specific gravity of the mucous membrane of the intestine, associated with characteristic morbid appearances, as shown by microscopic examination of sections. These combined modes of examination, not only in mucous membrane, but in all parts in which I have applied the test, have yielded one characteristic result—namely, that atrophic states of prolonged duration, which are generally described as *granular degenerations* of minute tissue, are, for the most part, of comparatively low specific gravity, and with a chemical re-action under the microscope, indicative of the presence of fat; while, on the contrary, the acute inflamma-

tory conditions of tissue (where loss of peculiar minute structures, like the glands of the intestines, is due to destruction from exudation and replacement by it) have uniformly high specific gravities. These results are similar to those which Dr. Bucknill has expressed with reference to the brain (*Med.-Chir. Review*, January, 1855, p. 212). A specific gravity of 1·032 to 1·033 of the mucous membrane, when it is free from congestion, may be regarded as associated with the healthy state of the gland-tissue. In conditions of *positive wasting* throughout, it descends to 1·030; while in conditions of *relative wasting*, where bulk is unchanged, but where glandular parts are displaced or destroyed by exudative deposits, the range of specific weight of parts examined has been as high as 1·044, and in the large intestines as high as 1·050. The specific gravity of Peyer's patches I have found to vary from 1·032 to 1·044, and even in patches from the same intestine there is often a considerable latitude in the range of the specific weights of the glands. The atrophic states of these glands are indicated—(1.) By a reticulated condition, void of all glandular elements, and which seems to occur naturally with the advance of years beyond forty; or to occur after extensive *infarction* during the progress of typhoid fever, the elimination of the product thus taking place without ulceration. (2.) By the remains of the cicatrices of ulceration in the form of dark granular deposits; or of a thin, clear, skin-like membrane, of a pale colour, and with a wrinkled contracted border. These morbid changes are most commonly associated with the prolonged continuance of complex morbid processes, as in constitutional diseases, such as those of anæmia (p. 56, *ante*), or in organic lesion leading to impairment of the constitution generally. They are also associated and expressed more or less locally amongst the peculiarly local glands or accumulations of glands of the gut, such as the solitary lenticular glands and the patches of Peyer, during the progress of some zymotic diseases, such as in cases of *cholera*, *dysentery*, and *typhoid fever*.

The definite morbid conditions in which the glandular apparatus may be found are as follows:—(1.) *Intumescence, stuffing*, or *cramming* of the gland, by different kinds of deposit, by debris of normal elements, or of both, and associated with redness and vascular congestion of the submucous tissue—conditions which correspond to the so-called "*infarction*" of the older pathologists;

(2.) *Softening, degeneration,* and *elimination* of the abnormal constituents in various ways; (3.) *Ulceration,* sometimes leading to sloughing, of whole patches of glands; (4.) *Collapse of solitary glands,* or otherwise cicatrized remains of ulcerations of solitary and aggregate glands; (5.) *Accumulation of melanic matter,* characteristic of pre-existent and long-continued vascular action, with changes in the tissue from hæmatine (*Pigmentary degeneration;* see vol. i., p. 117); (6.) *Amyloid degeneration* (see vol. i., p. 120).

The cramming of the glands, associated with submucous vascularity, is generally due to a milky-like exudation, with the variously metamorphosed epithelial elements. Such exudation may subsequently undergo various kinds of metamorphoses yet to be determined; but generally it may be stated that *corpuscular* and *fibrinous* elements co-exist in the various forms in which the glands are found intumescent.

There can be no doubt, from the observations of Dr. Handfield Jones, as well as from what is consistent with daily observation, that many of these obvious changes go on independently of any expressions of the phenomena of inflammation as commonly understood. But it is now well known also that the undoubted results of the inflammatory process sometimes occur without the manifestation of symptoms, such as *pleuritic effusions,* and which yield to appropriate remedies. In such cases the inflammation has been considered and described as latent. Does it not therefore appear consistent to associate the phenomena of such latent inflammations with the phenomena described by Goodsir and Redfern as the first stage of lesion in the cartilage-cell; with those of the early degeneration in the epithelium of the uriniferous tubes in Bright's disease; with the degeneration of involuntary muscle described by Quain; with the changes described by Virchow in the cornea, by Bucknill and Skae in the brain; with the early changes in the epithelium of the mucous surface of the bronchi, where they lose their cohesion, separate with abnormal rapidity, and ultimately assume forms and characters not to be distinguished from pus; and, lastly, with the phenomena of alteration and degeneration of the minute contents of these glandular parts now described; and consider such changes as expressions of one and the same complex morbid process, and which Virchow has described by the name of *parenchymatous inflammation?* (See vol. i., p. 99.) The ultimate results and

further expression of the inflammatory process varies, according to the texture of the part and other circumstances; but here we have the initiative of the process similar in all tissues, while its ultimate results are various; and while the complex state known as "fever" has found a constant expression in "the elevation of temperature," may we not be entitled to say that "the no less complex process of inflammation" has a no less constant expression in the diminished cohesion of the minute elements of tissue?

There seems to be conclusive evidence to show that much of the intestinal catarrh described by the common name of diarrhœa is associated with an *erythematous* congestion of the mucous surface of the lesser intestine, extending over a considerable extent, and rarely attended by increased arterial vascularity of the submucous tissue. When the symptoms of such congestive states are manifest during the progress of other diseases which terminate fatally, there may frequently be observed, besides the congested state of the mucous membrane, a marked increase of vascularity in other parts, such as the gastro-splenic omentum, mesentery, and glands, or infarction of the gastric glands, associated with congestion of the stomach generally. These phenomena for the most part are associated with a congested state of the hepatic system; and, occurring in a person otherwise in good health, give rise to symptoms which have been considered as a disease, and variously named *enteria, enteritis, erythemoidea, diarrhœa mucosa,* seu *catarrhosa* vel *catarrhale.* During the autumn and winter months in this country it is common to meet with such cases of disordered bowels in adults; and in children at any season, characterized by frequent fluid alvine discharges, and associated with extensive superficial irritation of the mucous surface. When the irritation predominates towards the upper part of the intestine—in the duodenum, for instance—the symptoms are an inclination to sickness, speedily followed by copious feculent discharges; the surface is easily affected by cold, and the individual may even shiver. There is also thirst, and a feeling of internal heat over the epigastric region. The functions of the liver are manifestly disordered at an early period, as expressed by the dull yellowish colour of the conjunctiva, and sallow darkness of the complexion, especially round the eyes. The tongue is generally moist, but viscid, clammy, and furred.

The appetite is completely lost, in the first instance. The skin is dry, and the palms of the hands and soles of the feet become unpleasantly hot and burning. The bowels generally become distended with flatus; and there is an uncomfortable sensation of distension, incapacity to expel the air, and occasional griping of the bowels, which are constantly producing a rumbling noise. The stools are at first large, feculent, and consistent, but subsequently they become watery, and even mixed with blood; then *tenesmus*, or a tendency to strain at stool, comes on and increases. Undigested articles of food are also passed — the characteristic symptom of *lientery* or *diarrhœa crapulosa* of the older authors. The belly is not painful when pressed, as in peritonitis or acute inflammation of the bowel, but there is often a deep-seated sense of uneasiness. This state soon terminates; in general, favourably. It is more especially brought on by exposure to great changes of temperature in humid and moist weather, by wet feet, damp beds or clothing, and improper dieting (after over-feeding, especially in summer weather) at irregular times; certain articles of food, imperfectly fermented malt liquors, acid wines, and sour unripe fruits, drastic purgatives, and various mineral poisons.

A diarrhœa of whitish stools is often extremely persistent, and may reduce the patient to the very verge of death. It may supervene after injuries which may induce cerebral concussion; or it may come on after dysenteries have been cured. There may be no fever, the appetite may be good, and digestion may seem tolerable, but emaciation and weakness become daily more and more marked. At first the motions only increase to two, in place of the usual single daily one. Afterwards the calls to stool increase, so that, during the twenty-four hours, there may be eight or ten in the day. The desire to evacuate the rectum becomes sudden; and the stools are apt to pass involuntarily, preceded by little or no premonitory sensations, and consisting merely of two or three table-spoonfuls of muco-gelatinous matter, resembling thick milk or puriform fluid, or like a jelly. A similar state of mucous membrane lining the small gut gives rise to a white secretion from its surface: so that white milky stools are observed to flow—*Diarrhœa alba* of Hilary—a form of bowel disease which is sometimes epidemic in Barbadoes. No remedy is of so much service in this affection as the *extract of nux vomica*, to the extent of *a fourth to a half grain dose;* or *strychnia* to the extent

of *one twelfth* of a grain, in a pill twice or thrice a day, with the *sulphate of iron* and *extract of calumba.* Dr. Maclean gave *tincture of the pernitrate of iron*, gr. x. every half hour, in this form of diarrhœa, with great benefit; and it is in diarrhœa of this kind that iron is of so much service. My attention has been recently called to a new preparation of iron by Mr. W. A. Moss (dispenser of medicines of the Army Hospital Corps at Dublin), which seems to possess some desirable properties, especially as to solubility and freedom from the inky astringent taste of preparations of iron. Its preparation is given in the note below.*

Black or dark stools (*melœna*) are not so much due to bile (*atrabilis* of Abernethy) as that such stools, resembling pitch, are principally composed of morbid or impaired secretions from the intestines (HOFFMAN, HOME, GRAVES). In such cases the discharge of the black matter is followed by a feeling of relief to the system generally. In cases of true *melœna*, where the dark colour is due to blood, great debility and sometimes fainting may follow the evacuations. Stimulating and tonic remedies, such as turpentine, are of benefit (GRAVES).

III. **Inflammation of the Bowel, or Enteritis.**—The phenomena of inflammation, when they do occur, generally express themselves in the ileum; and *Typhlitis* is understood to mean an inflammation of the cæcum.

Acute diffuse inflammation is marked by redness, thickening, and impaired cohesion. The redness is of a *deep venous red* approaching to blackness, either partial or general in extent, and in dotted, arborescent, or striated patches. It is distinguished from mere passive congestion by the increased arterial vascularity of the submucous tissue. The thickening is generally sensible, and often considerable. The impaired cohesion is not so obvious as in the stomach, but the mucous membrane may be removed much more easily than in health, from its attachment to the parts

* *Ferri Ammonia-Phosphas.*—Heat common phosphate of soda to redness. Take of the pyrophosphate of soda so obtained ℥ij. Dissolve in one pint of warm water. Then take of protosulphate of iron ℥iv. Dissolve in twelve ounces of water. Mix the solutions, collect, wash, and dry the precipitate at a gentle heat over a water-bath. Take of this precipitate ℥j., Liq. Ammonia P. L. ℥iss., water q. s. Dilute the Liq. Ammonia with an equal volume of water, and rub up with the phosphate of iron in a mortar until the latter is dissolved. Then dilute to ℥viij. Filter the solution, and evaporate at a heat not exceeding 120° Fahr., over a water-bath, and proceed as for the other scale preparations of iron. The dose is one fluid drachm.

subjacent. In the chronic forms of diffuse inflammation the colour, thickening, and cohesion of the gut are not greatly changed; but in general the thickness is more considerable, the cohesion of parts, instead of being impaired, is often rendered more tenacious, while the dark venous hue, on subsiding, leaves a greyish or slate-coloured tint, from a deposit of melanic matter in the substance of the membrane.

Serous inflammation of the mucous membrane of the small intestines may be inferred to exist from the large quantities of serous fluid often discharged by stool during life, at the same time that the abdomen is the seat of pain and tenderness. After death the fact may be proved by the loose diffluent fæcal matter often found in the small intestine: at the same time the mucous membrane is partially or generally inflamed.

The effusion of coagulable or fibrinous lymph in the small intestines is an extremely rare occurrence. "I have," says Dr. Baillie, "seen in violent inflammation scattered portions of coagulable lymph thrown out upon the surface of the villous membrane. This, however, is very uncommon" (p. 158). Billard has seen it but twice in the intestines of children. Dr. Handfield Jones also notices that the surface is sometimes the seat of an exudation much resembling that of croup; the attacks recurring several times, each presenting a stage of irritation, which ends in the formation and throwing off of a false membrane. This membrane sometimes forms a layer of some thickness, extending pretty uniformly over the surface, or appearing in the stools as tubular casts of the intestines, and sometimes it is as thin as a wafer, or consists merely of tattered shreds. In one case mentioned by Dr. Copland there were shreds of dysmenorrhœal false membrane discharged from the uterus, but not at the same time (*Pathological Anatomy*, by Jones and Sieveking, p. 526). The diphtheritic exudations described by Rokitansky are of a similar nature. Pseudo-membranous inflammations of the bowels have also been described by Dr. W. Cumming, of Edinburgh, and Dr. Simpson.

IV. **Ulceration** is much more common, and is indeed by no means unfrequent, especially from the action of typhoid and malaria poisons; and this ulceration may take place either at the free or adherent surface of the membrane. When it takes place at the free surface, the ulcer, says Andral, may form in the centre of a

point of inflammation, the mucous membrane around being healthy, or it may form in the midst of an extended patch of diffuse inflammation, without the follicular structure appearing to be in any degree affected. Again, the submucous tissue may inflame and become the seat of a number of small abscesses, which may point like so many pustules of small-pox. The apices of these abscesses become thinned and softened, till at length the mucous membrane ruptures, and the pus they contain is poured into the cavity of the intestine. These constitute miliary vesicles entirely of morbid origin from the first, and are to be distinguished from the infarction and ulceration of solitary or lenticular glands. The form, edge, and base of these ulcers are not unlike those found in the stomach, except that the ulcer with a sharp perpendicular edge, as if made by a punch, is much more rarely seen.

Besides *inflammation* and ulceration of the membrane over its general surface, the solitary glands may be either separately or conjointly with the former the seat of inflammation and of ulceration, subsequent collapse, atrophy, and obliteration.

The glands of the small intestines are thus liable to be enlarged and transparent, looking like a drop of pellucid water from serum, having a small black point in the centre, which is the mouth of the duct leading to the distended follicle. When the patches of Peyer are ulcerated, the ulcers generally takes the oval form of the patch, and these ulcers sometimes burrow so deep as to rupture the intestine. As these forms of lesion, however, principally occur in typhoid fever, dysentery, and cholera, as a result of the morbid process, the reader is referred to those articles.

Inflammation of the intestines, says Dr. Baillie, sometimes, although rarely, advances to mortification. When it does so, the mortified part assumes a dark livid colour, loses its tenacity, and is very readily torn.

Ulceration and mortification sometimes lead to the perforation or rupture of the intestine, when, the contents of the bowels escaping into the cavity of the abdomen, the patient dies of peritonitis.

The symptoms of *enteritis* partake more or less of those already stated as belonging to *intestinal catarrh*, combined with pain, aggravated by pressure, as a characteristic. The chief seat of pain is generally about the umbilicus, or the right iliac fossa. The discharges from the bowels relieve for the moment the griping pains. The pulse is excited, and generally full and strong.

The absence of intense pain and tenderness, of vomiting, of constipation, of excessive vomiting and tympanitis, of the small and frequent pulse, all of which are characteristic of peritonitis, is sufficient to distinguish enteritis from that disease.

V. Waxy or Lardaceous Degeneration of the Intestinal Mucous Membrane.—Next in frequency to the spleen, liver, and kidney degeneration of the lardaceous kind, is that of the intestines, where this degeneration especially affects the arterial capillaries of the villi, and the surrounding networks of the mucous and submucous tissue. Its progressive involvement of parts is as follows :—

It is seen—(1.) In the points of villi; (2.) Involving entire villi; (3.) In the mucous and submucous capillaries of inflamed parts; (4.) As annular infiltration round solitary glands; (5.) As degeneration of the vessels surrounding the sacculi of Peyer's patches. Virchow says he has seen the whole tract of arterial capillaries from the mouth to the anus in a uniform condition of amyloid degeneration. I have repeatedly met with this condition in dead soldiers at the invaliding hospital of the army, now at Netley. Anæmia of the mucous membrane, with a peculiar glistening or shining aspect of its surface, are the most characteristic features. Otherwise there are no outward signs of the lesion to attract attention. Pallor, anæmia, and atrophy ought to excite suspicion; but the application of the iodine re-agent is absolutely necessary. The atrophy has advanced so far that Virchow has known the villi to drop off, and the intestine to be bare of villi where villi are usually present. The walls of the fine arterial twigs of blood-vessels become pellucid, transparent, glistening, rigid, and thick, and a reduction in the size of their calibre is the result. Blood ceases to pass through them, nutrition is impaired, and atrophy results, extending over large tracts of the bowel,—most decided towards the duodenum. The substance of the villi has frequently been found changed into the lardaceous or albuminoid material. Sometimes the mucous membrane is also destroyed, and ulcers are developed which penetrate deeply into the tissue. The capsules of Peyer's patches have been seen enlarged, as well as the solitary glands (FRERICHS). Lambl has traced the degeneration and destruction of the intestinal epithelium through the substance of the villi, the follicles of Lieberkuhn, and the muscular coat itself.

Treatment.—The treatment of enteritis, when not arising from a morbid poison, is by leeches to the abdomen, gentle purgative medicines combined with an opiate, fomentations, and purgative or opiated enemata. After the inflammation has subsided, mild tonics, as the compound tincture of gentian with nitro-muriatic acid, may be substituted, with the prospect of recovering the lost tone of the parts.

When diarrhœa exists, if there is reason to believe that much congestion prevails connected with the hepatic region, a cathartic dose of calomel followed by castor oil may be necessary. If the stools indicate an acid re-action, *magnesia* may be given with advantage. When membranous films or shreds of coagulable lymph are passed, electro-galvanic applications over the abdomen and dorsal spinal region, combined with creosote or tar in the form of a pill, is quoted by Dr. Wood, on the authority of Drs. Cumming and Simpson, of Edinburgh, as worthy of a trial. Next to blood-letting are those remedies which determine towards the skin; and for this purpose combinations of Dover's with James's powder, or a solution of tartar emetic with laudanum, are the most useful medicines; and their beneficial action is very much aided by the use of the warm bath, and a flannel roller applied with firmness round the abdomen (CRAIGIE).

The greatest care ought to be given to the management of the diet. It ought to be strictly antiphlogistic, consisting entirely of slops and light puddings.

Animal food must be entirely withheld, and only permitted occasionally in the form of soup. The safest diets are those of arrow-root, sago, tapioca, gruel of oatmeal, *sowens*,* barley water,

* "The husk and some adhering starch separated from oats in the manufacture of oatmeal are sold in Scotland 'under the inconsistent name of *seeds*.' These, if infused in hot water, and allowed to become sourish in this state, yield, on expression, a mucilaginous liquid, which, on being sufficiently concentrated, forms a firm jelly known by the name of '*sowens*.' Not less than a quart of the *seeds* are to be rubbed for a considerable time with two quarts of hot water, after which the mixture is to be allowed to rest for several days till it become sour. It is then strained through a hair sieve, and the strained fluid left to rest till a white sediment subsides. The supernatant fluid is to be poured off, and the sediment washed with cold water; after which it may be either boiled with fresh water, stirring the whole time it is boiling, or it may be dried and prepared when convenient in the same manner as arrow-root. It may be eaten with wine, milk, or lemon juice and sugar" (Pereira *On Food*, p. 326).

toast water, burnt oatcake water. This last substance, used as a drink, is said to allay irritation and morbid sensibility of the bowels (CRAIGIE). When the abdominal pains and stools subside, and the appetite increases, the diet must be, if possible, still more scrupulously attended to. Oatmeal porridge boiled to the utmost possible degree of pulpiness, and not too consistent, is one of the best of diets, which may be alternated with ground rice, alone or combined with barley flour, all of which may be eaten with diluted milk from the cow, or with milk undiluted of town-fed cows.

VI. **Colic** is a painful affection of the lower portions of the abdomen, caused by a violent contraction of the muscular fibre of some portion of the intestinal canal. The remote causes are indigestion, exposure to cold, or other general cause, the effect of lead poison; and all periods of life, from infancy to old age, are liable to the affection. It also attacks either sex. It is seldom that persons die of colic; but such instances have occurred; and dissection has shown some portion of the intestines intus-suscepted —affording a strong presumption that this affection depends on a spasmodic constriction of some part of the intestinal canal. This view of the case is supported by Mr. Blane, who states that in fatal cases of colic in horses, different portions of the alimentary canal are found strongly contracted, and much oftener of the small than of the large intestines, which also sometimes contain gas. The bladder appears to participate in the spasm, the urine being either frequently ejected or suppressed. Colic, therefore, is a spasmodic contraction of the stomach or intestines,—usually sudden in its attack; and the patient consequently, without any previous indisposition, is often unexpectedly seized with a severe fixed pain in some part of the abdomen, but which is relieved on pressure, so that he either sits doubled up, or rolls on the ground, or lies flat on the belly. In other cases, where much air is secreted the bowels are greatly distended, and the pain is compared to a twisting or wringing around the navel, accompanied with soreness. The walls of the abdomen also participate in the internal spasm, so that the navel is often drawn in towards the back, or the heads of the *recti* muscles are exceedingly prominent, resembling so many round balls. The bowels are generally but not always constipated, and the stomach may or may not be irritable. In the latter case it often rejects both food and medicine. The pulse is little altered at the commencement of the attack; but if the

paroxysm be prolonged, and the patient exhausted by pain, it may be hurried and frequent. The tongue is generally clean, although sometimes white and coated.

Gastralgia, or stomach *colic*, is a severe pain in the stomach, often so completely idiopathic that the slightest cause produces it. One person cannot eat a strawberry, another a gooseberry, another an egg, without being seized with it. In other cases every sort of diet produces it, so that the patient is racked with pain after every meal. Those affected are usually adults; and women are more frequently the subject of it than men.

The attack of *colic* is generally sudden, the patient being unexpectedly seized with a pain, which attains its greatest height on the instant, round or above the umbilicus. This attack is generally accompanied by sickness or vomiting, by great flatulence, and by a confined or sometimes by a purged state of the bowels. It may last from a few minutes to a few hours, and often ceases as soon as the stomach is emptied or the bowels have acted; but when the patient is costive, it very constantly continues till he is relieved by medicine, when it subsides almost as rapidly as it commenced, leaving, however, a soreness behind it. The pulse in this affection is natural, there is no fever, and the pain is relieved on pressure—circumstances which readily distinguish it from inflammation. The disease may subside after one attack; but genuine gastralgia sometimes lasts for many months.

Diagnosis.—This disease is distinguished from inflammation by the pain being relieved on pressure, and by the quiet state of the pulse.

Prognosis is in every case favourable.

Treatment.—The treatment of colic is by opiates, chlorodyne stimulants, and purgative medicines. When the bowels are constipated, *five grains of calomel, fifteen grains of jalap*, and *one grain of opium* should be administered immediately, and followed by the following:—

℞. Mist. Camphoræ ℥ss. Magnesiæ Sulphat., ʒj.; Tinct. Hyoscyami, ♏xv. to xx.; Tinct. Cardamomi, ʒj. To be repeated every five or six hours until stools are obtained.

In mild cases a scruple of rhubarb, or half an ounce of castor oil, or other mild purgative, combined with a grain of opium, may be substituted for the opium, calomel, and jalap.

Some have doubted the propriety of administering opiates at the onset of the disease; but it is certain that a mild purgative, combined with a mild narcotic, will effect more than a drastic purgative without such combination. Enemata often give immediate relief. Externally, the application of large bags filled with hot *chamomile flowers*, or of *heated sand*, or of the *stomach-warmer filled with hot water, are useful.* The *warm bath fomentations*, or a *large lintseed* or *mustard poultice* over the abdomen, are also highly useful auxiliaries. Some patients are said, when these remedies have failed, to have been benefited by dashing cold water over the lower extremities; but the experiment is hazardous. The diet should, during the attack, be sago and arrow-root, with a little of brandy: and for some time after the patient has recovered it should be light, and perhaps limited to fish and puddings.

VII. **Obstruction** of the intestine occurs when some obstacle mechanically impedes the passage of contents through the bowel. A typical case is thus related by the late Dr. Brinton, whose monograph on this subject ought to be read by every student. He did more in his too short life to elucidate the pathology of such cases than any one who has written on this subject:—

"A person, perhaps hitherto healthy, experiences a sudden constipation, attended with disproportionate uneasiness, or flatulence, soon merging into pain and distension of the belly, with violent rolling movement of the intestines. The distension increasing, nausea and vomiting supervene; and gradually becoming more frequent, end by rejecting not merely any casual alimentary contents of the stomach, or the greenish, bilious, alkaline fluid commonly thrown up when this organ is unoccupied by food, but a fluid of greater opacity, colour, and consistence, with a distinctly fæcal odour. A further aggravation of these symptoms now conducts the malady to its termination. This, if fatal, is usually preceded locally by signs of paralysis, inflammation, or even rupture of the distended bowel, and constitutionally, by exhaustion or collapse replacing a febrile re-action. In other cases, the obstacle being removed by nature or art (if by the former, rarely before life is in extreme danger), the symptoms subside with comparative celerity. The pain, distension, and vomiting cease; the bowels are relieved by copious stools; and the patient (if not placed in further peril by any of those conditions incidental or consecutive to obstruction just hinted at) is rapidly restored to comparative health" (*Intestinal Obstruction*, p. 6).

The most remarkable and most characteristic symptom of intes-

tinal obstruction is the fæcal vomiting, explained by a doctrine that remained almost unquestioned since the time of Galen, until the observations and experiments of Dr. Brinton exposed the error, and established the pathology of intestinal obstruction on a rational basis. When, about twenty years ago, he exposed the error of the doctrine, then and even now entertained, it was supposed that fæcal vomiting was effected by an *anti*-peristaltic movement of the intestinal canal; that, at a certain stage of obstruction, the natural peristaltic action of the bowel above the occluded point was reversed: so that instead of proceeding towards the anus, as heretofore, it took the contrary direction, —thus impelling the intestinal contents in a similarly retrograde course, so as to return them to the stomach, whence they were vomited.

Dr. Brinton showed by abundant proof, experimental and incidental, that the notion of *anti*-peristalsis was contradicted by direct observation; and a careful study of the phenomena of intestinal obstruction, as witnessed in the human subject, and as artificially produced in experiments on animals, led him to the following theory:—

"The movement proper to the healthy intestine is a circular constriction or peristalsis, which, travelling slowly and intermittently down its muscular wall, propels its contents in a direction from the stomach to the anus. And when any part of the intestine has its cavity obliterated by an immovable mechanical obstacle, its contents, propelled by such a peristalsis, are stopped at the obstructed point. Here they gradually accumulate, so as first to fill and then to distend a variable length of the canal with a more or less liquid mass. But a peristalsis engaging the wall of a closed tube filled with liquid, and falling short of obliterating its calibre, sets up two currents in that liquid;—one at the surface or periphery of the tube, having the direction of the peristalsis itself, and one in its centre or axis, having

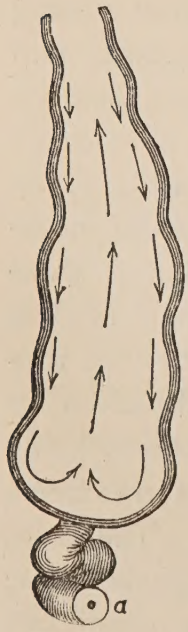

Fig. 44.

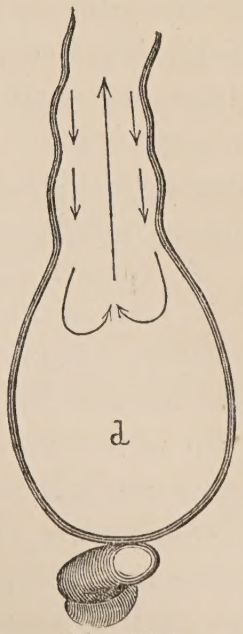

Fig. 45.*

*Figs. 44 and 45: Diagrams to illustrate the peristalsis of an obstructed bowel (after

precisely the reverse course. Those particles of the liquid which are in contact with the inner surface of the tube are propelled onwards by the muscular contraction of its wall. And this propulsion is necessarily accompanied by a backward current in those particles which occupy the axis or centre of the canal."

Fæcal vomiting is thus shown to result from the reflux of the intestinal peristalsis—a backward current in the liquids occupying the centre of the tube.

The ordinary course of intestinal obstruction from Dr. Brinton's point of view divides itself into two stages. In the first stage the healthy actions of the bowel are continued; in the second stage they are arrested or utterly and permanently annihilated by paralysis, enteritis, or peritonitis.

In the first stage, abnormal *distension* of the intestine can generally be felt through the yielding wall of the belly—as a condition of fæcal vomiting—from the very commencement, and continuing a persistent physical sign through all the stages of the obstruction. Experience led Dr. Brinton to assert "that the accumulation of intestinal contents immediately above the obstructed point may sometimes be detected as a slight fulness to palpation, and a much more definite dulness to percussion, where many of the other indications of obstruction are scarcely perceptible, or even absent." The movements of the obstructed intestine may thus be traced rising visibly against the walls of the belly covering the obstructed tube, "in coils that may be fancifully compared to those of a writhing serpent," until the paralysis and collapse usher in the fatal issue.

The character of the pain in obstruction is variable. It is sometimes sudden and violent, and often rises to great intensity in a very short time. It is distinct from the burning pain of peritonitis. It is usually intense in intus-susception, and in the impaction of gall-stones; somewhat less marked in the obstruction produced by twisting of the bowel, or by bands and adhesions; scanty in the obstruction of stricture; and almost absent in the obstruction caused by the impaction of fæces in the large intestine (BRINTON).

DR. BRINTON).—Fig. 44. Stage of moderate distension, with forward and backward currents, as indicated by the arrows traversing the whole tube above the obstacle; (*a*) Contracted segment of intestine below the obstacle. Fig. 45. Stage of extreme distension, in which (*d*) the dilated and paralyzed segment above the obstacle is scarcely engaged by either of these currents.

Exclusive of hernia, Dr. Brinton has estimated, from an analysis of 12,000 necropsies, that obstructions of the intestine cause about 1 in every 280 deaths from all diseases indifferently; and the chief varieties of obstructions have to each other the following proportionate frequency:—Intus-susceptions or invaginations, 43 *per cent.;* obstructions by bands, adhesions, diverticula, or peritoneum, external to the bowel, 31½ *per cent.;* strictures (including a few tumors) involving the intestinal wall, 17½ *per cent.;* torsion of the bowel on its axis, 8 *per cent.*

The forms of intestinal obstruction are mainly as follows:—

(a.) Intus-susception, of which the varieties are,—*Ileo-cæcal,* 56 *per cent.; Iliac,* 28 *per cent.; Jejunal,* 6 *per cent.; Colic,* 12 *per cent.*

(b.) *Obstructions due to bands, adhesions, diverticula, gall-stones, lesions,* such as rupture of *mesentery,* and other peritoneal lesions. The small intestine is the seat of the obstacle in 94·53 cases *per cent.*

(c.) *Obstruction due to strictures, tumors, or twistings of the bowel and mesentery,* give about 87·36 *per cent.* of cases involving the large intestine.

Intus-susception is the accidental insertion or protrusion of an upper into a lower segment of intestine. It occurs more frequently in infancy and childhood than at any other period of life. Of twenty-five cases observed or collected by Rilliet, *seven* occurred in children of six months or under; *six* during the first year of life; *seven* between five and ten years of age; *five* between ten and under fifteen years of age. Dr. Brinton's experience goes to show that half the ileo-cæcal intus-susceptions are infants under seven years of age; many but a few months old; and he gives the average ages of the ileo-cæcal, jejunal, and colic respectively as 18·57, 34·6, and 31·4 years.

Invaginations of the small intestines are so frequently found after death, in comparatively young and well nourished subjects, that it is generally believed they are formed with great facility, and that they often occur during life, giving rise to temporary bowel derangement; but that they also soon become disentangled again by the normal peristaltic movements. Of 300 children examined by Louis at the Salpetrière Hospital, and who died there, the greater number had two, three, or more *volvuli* without any inflammation of the parts; and there were no circumstances

in their history during life which led to the suspicion that these children suffered from intus-susception, (*Mem. de l'Acad,* vol. iv.) Dr. Baillie, also, in his great work *On Morbid Anatomy,* says, "In opening bodies, particularly of infants, an intus-susception is not unfrequently found which had been attended by no mischief; the parts appear perfectly free from inflammation, and they would probably have been easily disentangled from each other by their natural peristaltic motion." Dr. Macintosh states that he scarcely ever opened a child without finding partial invagination of the small intestines (*Practice of Physic,* vol. i., p. 256). Dr. Hodgkin, in his valuable work *On the Morbid Anatomy of the Mucous Membranes,* and Rokitansky in his *Pathological Anatomy,* each make mention of the frequent occurrence of invaginations in the bodies of adults as well as of children, and they consider them to be produced in the majority of instances during the last moments of life—in the death-struggle, or in the *rigor mortis* of the dead intestine. I have frequently observed such invaginations in *post-mortem* examinations, not associated with symptoms during life, and easily reduced by traction; but they have been generally in cases where the irritability of the bowel had been greatly increased during life by excessive diarrhœa, with or without ulcerations of the intestines. Such invaginations were invariably in the small intestines.

Cases of intus-susception *in the adult* are rare—so rare, indeed, that in the extensive experience of one of the largest civil hospitals in London (Guy's), Dr. Wilks records that "he has never seen but one case of intus-susception *in an adult,* and in this case the obstruction was never complete, and death did not occur for some weeks" (*Pathological Anatomy,* p. 292). In the *Transactions of the Pathological Society of London,* extending over the first fifteen years of its existence, there are only seven cases of intus-susception *in the adult* on record—no two of which occurred in the individual experience of any one man. The ages of these seven cases are respectively as follows—namely, eighteen, twenty-five, thirty-two, thirty-four, forty-one, and two at forty-four years of age. In one case the symptoms continued for three months, and at last ended in recovery after the passage of a portion of ileum (*containing a polypoid tumor*) by the rectum. In another case the symptoms continued during four months, and terminated fatally by exhaustion. In my own experience I have never made a *post-*

mortem examination of a case of intus-susception *in an adult*, nor have I ever seen a case of intus-susception *in an adult* during life. In the Museum of the Army Medical Department at Netley there are preparations showing the lesions and morbid relations of the parts preserved, from at least eight cases occurring in soldiers at ages varying from twenty to forty-two years of age.

A case recorded by Dr. Todd, in the *Army Reports for* 1864, p. 532, is of great interest, inasmuch as the dissection of the parts, made by me at Netley, shows that the intus-susception was associated with a large polypus growing from the mucous surface of the small intestine. The history of the case further shows that it was preceded and accompanied by intense and severe diarrhœa; and in the course of examination of the parts sent to Netley, my coadjutor, Dr. Davidson, discovered that the mucous membrane of the *caput cœcum* was infested by the minute parasite known as the *trichocephalus dispar*. This parasite is a very minute round worm, with its head-end of hair-like fineness, usually firmly fixed to the mucous membrane of the intestines, while the rest of the body is generally coiled upon itself and hidden amongst the mucous secretion of the gut. The natural history of this entozoön shows that it has oftentimes been associated with severe epidemics of diarrhœa. Indeed, its discovery more than 100 years ago (1760-61) was made during the prevalence of a severe epidemic of diarrhœa (*morbus mucosus*) amongst the soldiers of the French army, associated with the presence of this parasite in the *caput cœcum* of those who died of the disease.

Five cases of intus-susception are recorded in the *Transactions of the Pathological Society* which are associated with polypoid tumors of the intestine at or near the site of lesion. The latter case referred to in these *Transactions* terminated favourably after the passage of the invaginated portion of gut (containing the polypus), the symptoms having continued for three months.

The *volvulus*, or portion of the gut where the obstruction exists, consists—(1.) Of the external portion formed by that part of the bowel into which the other has slipped ; (2.) Of the middle; and (3.) Of the internal part, composed of the reflection of the invaginated portions. As it is not always easy to follow the anatomical relations of the several layers of structures composing an intus-susception, it may be of use to give a diagramatic outline of the relation of the serous and mucous coats of the intestine

in such lesions; because, it is of practical importance to remember that, although the parts are greatly displaced, yet the anatomical relations of the serous and mucous surfaces of the intestine are never altered. Textures of the same anatomical character are always in contact one with another, and the channel of the gut along its mucous surface is always open. That such is the case may be understood by taking the leg of a long stocking from which the toe-end has been cut off, so that the stocking may be converted into a continuous tube open at both ends. If one portion of the stocking be then drawn into the other, a correct imitation of the relation of surfaces in an *intus-susception* will be obtained.

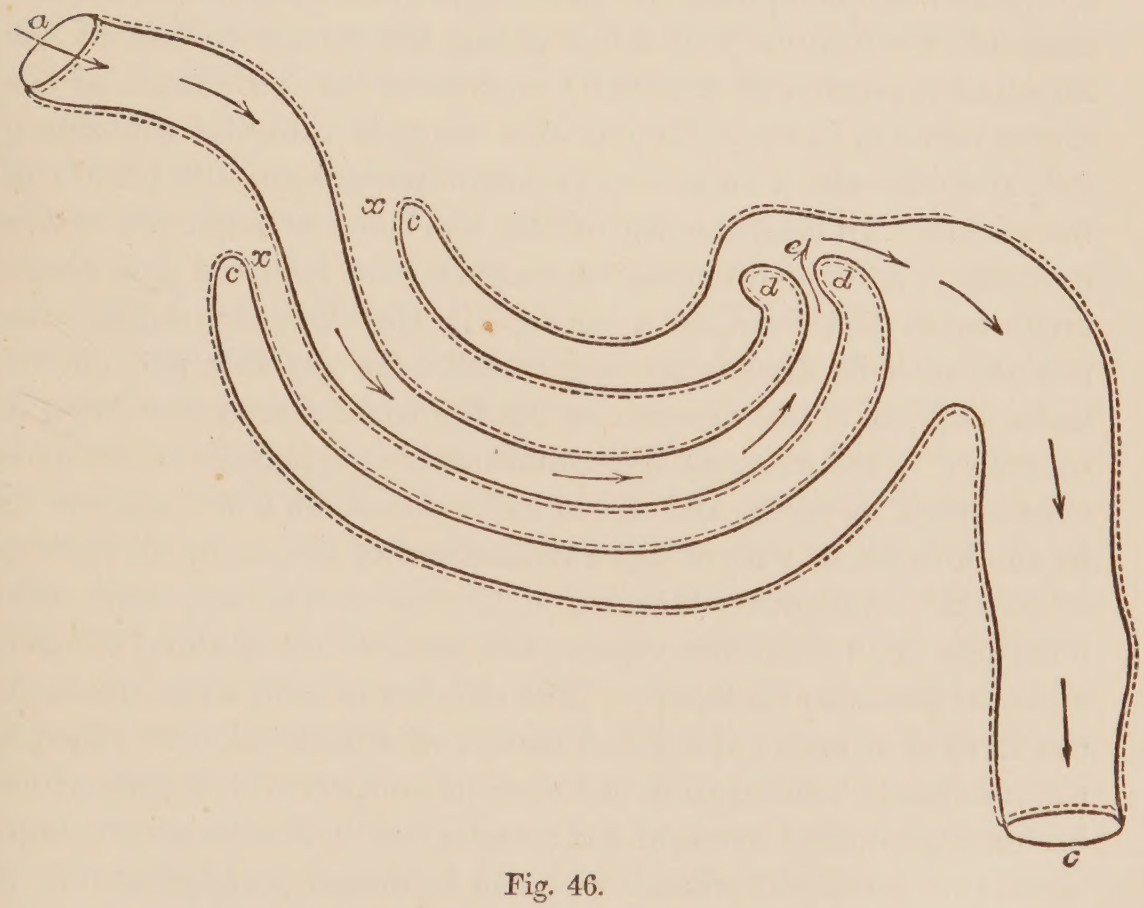

Fig. 46.

In the diagram (fig. 46) the tube, *a b*, may be traced to be continuous, as indicated by the arrows. The dotted line is meant to correspond to the *serous* surface, and the thick dark line to represent the *mucous* surface of an intestine comprehending an *intus-susception.* From the outer to the *innermost* surface at the site of lesion, on cutting through one layer, *the first* of the enclosing gut, a MUCOUS SURFACE is reached, which has a *cul de sac* reflection

at *c*. Thus, the MUCOUS surfaces of the including and the included portions of intestine are in constant apposition, rubbing against each other. On cutting through the *second* layer of the *intus-susception*, a SEROUS SURFACE is reached which has a *cul de sac* reflection at *d*. Thus, the SEROUS surfaces of the includ*ing* and the includ*ed* portions of intestine are in constant apposition, rubbing against each other. At *e* the mucous canal is always more or less open in cases of simple *intus-susception;* but the orifice is invariably turned or curved to one side, and may be so firmly applied against the mucous surface of the including intestine (by the dragging of the mesentery which has been included) that the orifice may be closed, like a valve, by simple apposition and compression. In consequence of the lateral attachment of the mesentery to a line along the serous surface of the intestine, a portion of mesentery equivalent to the extent of the serous surfaces in apposition is also dragged into the containing gut, and exercises a most important influence upon the nature of the lesions. In consequence of the one-sided attachment of the mesentery, and the dragging of its parts, the included gut necessarily takes the form of a curve. It thus appears highly corrugated over its mucous surface, dragged to one side and curved upon itself, as it lies exposed on cutting up the outermost layer of intestine. The orifice of the contained or invaginated intestine is thus turned upwards, and is not to be found at what appears to be the lowermost part of the extreme end of the included portion of bowel. This great dragging of the mesentery necessarily also obstructs the mesenteric vessels, and leads to the gradual effusion of blood between its layers. This effusion is seen after death in the form of compact indurated masses of a dark colour. Blood is also gradually effused from the mucous surface of the gut, which becomes gradually strangulated; and, combined with other symptoms, this persistent effusion of blood is almost pathognomonic of incarceration of a bowel in the form of intus-susception.

In some cases the pressure of the inclosed bowel on the containing gut, and the dragging of the included mesentery, are so intense, that the invaginated portion has actually effected an opening by ulceration through the inclosing bowel, and so projected into the cavity of the peritoneum, before death took place. In such cases the fatal result was generally by peritonitis. Four specimens in the Museum of the Army Medical Department illus-

trate this fact in the pathology of intus-susception. One has no history; another is quoted as an example of the bad effects of purgation; the third is from a woman aged twenty-four, who had been ill ten days, when an extensive opening occurred through the inclosing bowel, and peritonitis of a severe form speedily proved fatal. A fourth occurred in a soldier aged twenty-two. In this specimen a very large mass of gut is involved, and it is very significant of the injurious influence of purgation in such cases. Although this soldier is recorded to have had persistent diarrhœa, flatulence, bloody stools, and other symptoms of intus-susception, he was nevertheless alleged to be a malingerer. He was treated with purgatives, and lived long enough for the end of the included gut to wear a hole, by pressure and rubbing, through the substance of the containing bowel. No one can doubt but that such a lesion would be greatly aggravated by purgative remedies.

In all cases of intus-susception, whether occurring in children or in adults, the administration of purgative medicines tends to aggravate the lesion and the symptoms. Accordingly the rule of practice is absolute—namely, "to withhold all purgative medicines from the commencement in cases of intus-susception." The bowel being incarcerated, the stimulus of purgation proceeding from above downwards is quite unable to undo the incarceration of an intus-susception. A purgative, therefore, acts injuriously as a stimulus which cannot be obeyed; and the obvious tendency of a purgative is to increase the peristaltic action of the bowels, and therefore to increase still more the invagination. That such is the case will be readily understood by a physiological consideration of the phenomena of intus-susception; for although it is not always easy to account for the first beginning of an invagination, yet physiology enables us to understand how, an invagination once begun, the lesion tends to increase—(1.) From the peristaltic action of the bowel, greatly stimulated and increased by irritation of every kind, so long as tonic irritability continues; (2.) From the spasmodic action of the part of the gut *above* the invagination preventing spontaneous return; (3.) From the invagination being thus completed, it continues permanent, tenesmus occurs, and thus the violent and repeated contractions of the abdominal muscles tend still more to maintain and increase the lesion. The constant motion and pressure of parts one upon another in some cases is so great, that the end of the invagination has been known to pene-

trate through the walls of the inclosing bowel, so as to appear in the cavity of the peritoneum.

The injurious tendency of purgative medicines will be also still more apparent if a lesson is taken from the teachings of morbid anatomy. *Post-mortem* examination, combined with a study of the phenomena of intus-susception during life, shows that the increase of the lesion takes place mainly at the expense of the *external containing* portion of the bowel; and therefore, also, it can readily be understood how some fixed point in the bowel is the first starting-point of an invagination. Most frequently (56 *per cent.*, BRINTON) it is the ileum and cæcum which pass into the colon, then the colon passes into itself, so that the *appendix vermiformis cœci* becomes included. Two orifices then exist at the extreme end of the invagination:—one is that of the lesser bowel, the ileo-cæcal valve, the other is the entrance into the *appendix cœci*. Two preparations in the Museum of the Army Medical Department show this arrangement of the parts composing the lesion in cases of invagination. In 32 *per cent.* (BRINTON) the small intestine forms all the layers. In 12 *per cent.* (BRINTON) the colon is exclusively involved; and the rectum scarcely ever forms more than the outer layer.

Causes.—In all the dissections of invagination whose history I have examined, they have either been associated with the diarrhœa of irritation (as from worms, undigested masses of food); or with cerebral lesions (as in the cases of children in whom invaginations are very common); or with ulcers of the intestines, or polypoid growths.

In giving a summary of cases, Dr. Peacock finds that, while in some cases no cause could be assigned for the affection, in others the disease appeared to have been excited by accidents, taking injudiciously large meals or improper food, by the irritation of drastic purgatives, or the presence of worms. Of the cases which he analyzed, "in one instance the disease followed a kick, in another the carrying of a heavy weight, and in a third the taking of a large meal; in one an active purgative had been taken a few days previously, and in one there were worms in the bowels." In the case he particularly records to the Society, "the predisposing cause might possibly be the small polypus which was found attached to the mucous membrane, near what appeared to be the upper end of the invaginated portion" (*Trans. Path. Soc.*, vol. xv., p. 117).

As to how the lesion first commences some notion may be obtained, and the physiology of the process may, to some extent, be comprehended by experiments on the intestines of animals while under chloroform, or just after having been killed. If a portion of the small intestine be pinched with a pair of forceps, active circular contraction and constriction of the gut immediately commences at the site of irritation. This constriction continues for some time, and is transmitted, or advances onwards, under the influence of the usual peristaltic action of the intestines. Wave upon wave of constrictions may be made in this way to follow each other in succession, so long as the vital irritability of the intestine continues. If the advance of the constriction onwards is impeded by any cause, such for instance as an undigested mass of food, a scybalous portion of fæces, a foreign body, or a polypoid growth, or even another constriction, and if the onward motion of the bowel fails to dislodge the obstruction, a partial invagination very readily occurs; but where the obstruction is necessarily localized (as from ulcers, polypoid growths, or fixed parasites) permanent invagination commencing in the vicinity of such local lesions is more readily induced. The mere weight of a polypoid growth would necessarily favour the occurrence of invagination by dragging down the bowel to which it is attached, and so inverting its coats. In 5 *per cent* (BRINTON) the intus-susception is so caused; and often situated above the ileo-colic valve, the polypus having made its way through it, the spasmodic contraction of the valve would effectually prevent the spontaneous return of the invagination.

Another efficient and increasing cause of obstruction exists in the included mesentery. By its inclusion it causes such a drag upon the included portion, that the mucous surface of the bowel is not only greatly corrugated, but by apposition against the containing bowel the narrow canal through the invagination becomes completely closed. The anatomy of the parts at once shows how this is effected. The mesentery being attached only to one side of the gut, it drags the intestine to that side; and the greater the amount of bowel invaginated, the greater will be the curvature of the included portion upon the mesentery as an axis; and the greater and earlier will be the complete obstruction.

Thus, when a case of intus-susception is examined after death, the invaginated portion of bowel is always seen in the form of a

curve lying within the including intestine. The extreme end of the invaginated portion thus comes to be so turned upon itself that the canal by apposition is completely shut up. The blood-vessels of the impacted mesentery also undergo great and increasing congestion; and as strangulation and obstruction become complete, indurated masses of blood may be found effused within the folds of the mesentery. As strangulation increases, blood exudes from the mucous surface of the intestine, so that small flocculi of blood, as well as fluid blood mixed with mucus and free from fæcal matter, continue to pass *per rectum* as long as the canal remains open, and such symptoms (hæmorrhoids excluded) are justly regarded as pathognomonic of incarceration of the bowel from a simple intus-susception not yet completely closed.

Symptoms of Intus-susception.—The symptoms indicate obstruction and inflammation. *In the child* they mainly consist of restlessness, sudden fits of crying, and straining as if at stool; a discharge of mucus, more or less mixed with blood and free from fæcal matter, sickness, and anxiety of countenance. These phenomena are generally, but not invariably, preceded by a sudden and violent action of the bowels. A physical examination of the belly may disclose a tumor or swelling of the intestine; and sometimes, as in a case described by my friend and neighbour, Dr Orsborne, of Bitterne, to the Medical Society of Southampton, the invaginated part could be reached with the finger introduced into the rectum.

In the adult the symptoms and phenomena in cases of complete strangulation of the gut by strictures, like internal strangulation from bands of lymph, or twisting of the gut round such constrictions, are sudden; and if the stricture be not relieved, the case proves fatal about the fifth or sixth day. On the other hand, in cases of intus-susception the symptoms of the incarceration are by no means sudden nor rapid in their progress; or, rather, they are comparatively slower in their development and progress to a fatal issue than cases of complete obstruction by strangulation are.

In cases of intus-susception the impediment from the first is partial, and in some cases the obstruction is never complete, although the symptoms may extend over many days, or weeks, or even months. One case is on record in the *Transactions of the Pathological Society* in which the symptoms of incarceration

were present during four months, and although adhesions had formed between the serous coats of the invagination, yet obstruction of the intestinal canal was never complete.

A summary of the prominent phenomena of intus-susception may be stated as follows, showing the comparative slowness and incompleteness of the obstruction, as well as bringing forward those phenomena which (when weighed with other symptoms) may be regarded as pathognomonic of intus-susception. Diarrhœa early is generally the first indication of illness. In connection with this diarrhœa and its prevalence, the existence of parasites must not be overlooked, inasmuch as their existence in one patient renders it probable that they may exist also in others, as a cause of the prevalence of diarrhœa in an epidemic form. Collapse, nausea, and diarrhœa, are the next symptoms to supervene, followed by tenesmus, or a feeling of fulness in the rectum; and after going two or three times to stool, *blood only* is observed *to pass*, and similar discharges continue. An enormous discharge of fæces may sometimes take place after an enema, but generally accompanied by *a large flow of blood.* So long as the patient lives *he continues to pass blood at stool*, and on occasions scybala. Small portions of hardened fæces may continue to be got rid of, but every effort is attended by *the usual flow of blood.* Purgative remedies aggravate the symptoms. Death is comparatively slow; and hence the extensive coagula which are found in the cavities of the heart.

Diagnosis is therefore mainly differential between this and other forms of alvine obstruction. The colic is extremely severe, and the marked remissions of pain are followed by exacerbations, which increase in violence with each repetition. Physical diagnosis ought to be had recourse to in every case. From hernia, cases of intus-susception are to be distinguished by the absence of the hernial tumor at the respective abdominal apertures where hernia is usual; but a tumor within the abdomen is a physical sign of the greatest value; and is not often absent, though easily overlooked, as it is often of small size (BRINTON). The condition of the rectum ought always to be ascertained by a digital examination. The vomiting is less urgent in proportion as the obstruction is lower down in the bowel; if the duodenum is involved, vomiting is almost incessant. Dr. Orsborne, of Bitterne, lately brought me the stomach and intestines of a child that died of this

affection; and the relations of the parts involved were peculiar in this respect, that in the reflection of the colon, which contained the volvulus, the mesentery of the duodenum was dragged in, and along with it the adjacent wall of that intestine was also taken in. The parts are preserved in the museum at Netley.

It is necessary to distinguish between ileo-cœcal, or the colic invagination, and that of the small intestine. The former is distinguished from the latter by—

"(1.) The prominence of tenesmus, which is rarely present in any marked degree, where the small intestine only is implicated; (2.) The greater size and fixation, as well as the different site of the tumor, which, if large, generally proceeds towards the left side of the hypogastric or left iliac region; (3.) The subordinate share taken by hæmorrhage, which, instead of copious bleeding by stool and vomit, is often little more than a scanty admixture, scarcely sufficient to tinge the mucus passed from the bowels, with violent and frequent straining by the patient; (4.) The still more subordinate share taken by obstruction, which not only seems to be often anticipated by death, as regards any complete symptoms of its presence, but to be really absent, owing to the patulous state of the axis of the invagination; (5.) The presence of the end of the invagination in the rectum" (Brinton).

Obstruction of the small intestine is chiefly characterized by (1.) The umbilical seat of the pain, which is also more early and severe; (2.) Vomiting is more early, severe, and frequent; obstruction is much more rapid, constant, and complete in the small than in the large intestine.

Prognosis is not always hopeless, but is nevertheless most grave. The cases which recover are almost invariably chronic or protracted ones. In the fifteenth volume of the *Transactions of the Pathological Society of London*, Dr. Peacock gives an account of the passage of a large piece of bowel by the rectum in a case of invagination, followed by recovery; and in that paper he refers to eighty-eight cases of intus-susception in which portions of bowel are reported to have passed by the rectum. Those which end by expulsion of the *volvulus* have a duration of from twice to thrice as long as that of the fatal cases; and it is not till the second, third, or even sixth week that the remission of symptoms occurs which announces the relief of the obstruction, and which often precedes by a day or two the first healthy alvine evacuations.

It is to be remembered that distension of the involved bowel measures not only the danger, but the rapidity of the fatal issue.

The cases of most serious import are those which, commencing in the small intestine, involve the ileo-colic valve; and the majority of fatal cases are those in which the cæcum and ascending colon have swallowed up, as it were, a large portion of the small intestine. The danger may not be at first appreciated, because the obstruction is never complete in the first instance, as we know from the nature of the lesion as well as from the accurate history of cases. The bowel, at first, is merely incarcerated by the invagination, and it is not until the middle and internal portions, with their contained mesentery, become compressed, constricted, and ultimately strangulated, that complete obstruction ensues. Till this occurs, the continuous expulsion of bloody mucus from the central tube of the inversion goes on. Inflammation and sloughing thus commence as the parts become subjected to more and more increasing pressure.

Two forms of inflammation prevail — namely, one, serous, between the opposed peritoneal surfaces, and commencing at the angle of reflection of the middle on the external layer (*x*, *x*, *x*, in the diagram, p. 863), at the part where the one portion slips into the other. It is here that the peritoneal surfaces of the invagination commence to adhere; and up to the period of adhesion the ancient remedy inculcated by Hippocrates, of injecting air into the great gut by a long tube, introduced *per rectum*, has effected the greatest number of cures—forcing, by gentle, persistent, and equal pressure, the invagination backwards, and so causing it to be undone. The other form of inflammation takes place between the two opposed mucous surfaces (*c*, *c*, in the diagram). At this angle of reflection an abundant white leucorrhœal-like secretion (corpuscular elements of inflammation with mucin) commences very early to be discharged by the rubbing of the opposed surfaces, and eventually the inflammation (mucous and serous) may be so destructive that ulceration and sloughing of the whole invagination may be the consequence. In this way continuity of the canal may be restored—the invagination having passed, *per rectum*, as a slough. Dr. Brinton believes this favourable termination occurs in not less than one in every two or three cases of intus-susception. On an average, separation of the slough is not complete before the eighth day, and the liberated

bowel is rarely expelled *per anum* before the tenth or twelfth day, and is sometimes only prevented by the death of the patient on the fifth or sixth day. Of such cases eighty-eight are now on record, an analysis of which, by Dr. Peacock, has been already referred to (*Path. Soc. Trans.*, vol. xv., p. 114).

Treatment.—The following are the details of treatment for cases of intus-susception advocated by the late Dr. Brinton. The chief indications and treatment are—(1.) *To prevent distension,* by reducing, in every possible way, the quantity of food and drink, restricting the latter to small but frequent sips (preferably through a long straw or tube) of cool, iced liquids. Food is to be given, as strong beef-tea, soup, or milk, with equal frequency and caution. Small doses of alcohol (as brandy with water, or soda-water) is to be alternated. But if any repugnance exist to food or drink, or if vomiting is excited by these articles, the amount given must be reduced. Water, milk, and gruel are to be given freely in often-repeated enemata.

(2.) *To assuage pain and to mitigate excessive peristalsis* suggest the same kind of remedy—namely, *opium,* which is to be given continuously and alone, in the solid form—preferably in the *extract.* The practical limit of the dose is indicated by the comparative arrest of pain, the approach of narcotism, and decided contraction of the pupil.

Belladonna is only of use as a remedy to diminish the straining peristalsis. It may be given *combined with opium,* as *two* parts of *extract of opium* to one, two-thirds, or even one-half part of *extract of belladonna* in a pill.

Enemata are useful as a mechanical aid to removing obstruction. They may gradually distend the bowel at the site of obstruction, so as to effect such a change in its position and arrangement as may release the impacted portion. The administration of enemata is only safe and efficient if undertaken by a person of competent skill. The quantity of fluid must be injected little by little, and must be retained as long as possible; and the patient must resolve to tolerate some pain in reaching that climax of distension at which only enemata are calculated to relieve obstruction.

In cases where the intus-susception is in the large intestine, inflation of the bowel with air, as originally suggested by Hippocrates two thousand years ago, in his third book (Περι

Παθων), has of late years been revived and adopted, first in America, and subsequently in this country, (Gorham, in *Guy's Hosp. Reports*, and *Med.-Chir. Trans.*, vol. ix.) This Hippocratic remedy has undoubtedly been more successful than any other. Of twenty-eight cases, the details of which were collected by Dr. Orsborne, there were only *seven* recoveries; and three of these were effected by inflation of the colon with air. Dr. Murphy and Mr. Erichson have borne strong testimony to its value in discussions on the subject; and the former had recourse to the operation on one of his own children with success (MS. notes of Dr. Orsborne). For its success, Dr. Orsborne is of opinion that the remedy should be employed at an early period, before there has been time for adhesion between the contiguous surface of the volvulus. My friend Dr. David Greig, of Dundee, has had recourse to this method of treatment in numerous instances with perfect success, and has published an account of his experience in the *Edin. Monthly Med. Journal* for October, 1864. By means of the ordinary elastic enema tube, fitted to the pipe of a small pair of bellows, he was able to pass a considerable quantity of air into the rectum, continuing the process till the belly shows signs of considerable distension, and even till uneasiness prevails. At the same time warm fomentations are to be applied to the belly. Its beneficial action is indicated by the relief of the urgent symptoms, such as straining and vomiting; and gradually a fæcal evacuation is obtained from the bowels. The use of large enemata, with manipulation, has also been recommended. A long stomach-tube is to be passed as high up the colon as it will go, and the anus being firmly compressed round it, warm water is to be slowly injected, so as to distend the bowel as much as possible. When the fluid is allowed to come away, the abdomen should be pressed upon with the hands, so as to move about the coils of intestine (Tanner).

The earlier any of these remedies are had recourse to, the greater will be the chance of recovery; and the patient may be put under the influence of chloroform to facilitate the manipulations. When all remedies fail, gastrotomy may be thought of, and its chances of success considered. The operation is advocated by Benjamin Phillips, and in some cases it may be justifiable, (see his paper in *Med.-Chir. Trans.*, vol. xxxi.) The

only cases for which it seems suitable are in obstructions from bands, diverticula, and the like lesions affecting the small intestine. The object of the operation is to divide the cord-like cause of strangulation (BRINTON).

SECTION III.—RELATIVE WEIGHT OF THE SOLID VISCERA OF THE ABDOMEN.

The liver, the kidneys, the spleen, and the pancreas are the organs which require special notice here; and in order to obtain positive data for the solution of many questions regarding their pathology, it is necessary to know the relative weight of these viscera.

TABLE SHOWING THE RELATIVE AVERAGES OF BODY-WEIGHT, AND THE WEIGHT OF THE SOLID VISCERA OF THE ABDOMEN AS TO AGE AND HEIGHT (BOYD).

Age and Sex.	Body-weight.		Body-height.	Weight of Liver.	Weight of Spleen.	Weight of Pancreas.	Weight of Kidneys.
Years.	Lbs.	Oz.	Inches.	Ounces.	Ounces.	Ounces.	Ounces.
1 to 2 {Male,.....	14	6	28·5	11·7	1·34	·44	2·65
1 to 2 {Female,...	13	2	27·7	11·17	1·04	·49	2·4
2 to 4 {Male,.....	20	0	31·6	16·85	1·58	·76	3 33
2 to 4 {Female,...	18	7 5	31·6	13·49	1·28	·68	3 14
4 to 7 {Male,.....	25	8	37·5	19 13	1·85	·8	4·05
4 to 7 {Female,...	24	9	37·0	19·56	1·65	·8	4·26
7 to 14 {Male,.....	42	6	47·0	34·71	3·03	1·68	6·58
7 to 14 {Female,...	38	6	45·0	25·86	2·54	1·34	5·75
14 to 20 {Male,.....	68	0	60·5	57·76	5·19	2·19	9·34
14 to 20 {Female,...	63	14	57·7	54·33	4·68	2·64	9·09
20 to 30 {Male,.....	92	14·5	66·7	60·29	7·19	3·54	11·57
20 to 30 {Female,...	86	13	62·0	52·74	6·53	2·95	10·17
30 to 40 {Male,.....	98	3·5	66·5	58·11	7·12	3·47	11·35
30 to 40 {Female,...	87	0	62·0	53·61	6·13	3·05	10·34
40 to 50 {Male,.....	102	0	66·8	58·06	6·19	3·48	10 89
40 to 50 {Female,...	84	9·5	62·0	49·03	5·04	2 73	8·8
50 to 60 {Male,.....	102	0·5	66·0	55·37	6·23	3·46	9·1
50 to 60 {Female,...	86	0	62·0	44·0	4·67	2·83	8·55
60 to 70 {Male,.....	103	13	65·7	48·24	4·82	3·1	8·83
60 to 70 {Female,...	86	14	61·5	42·98	4·07	2·8	8·28
70 to 80 {Male,.....	106	13	65·7	46·33	4·93	3·22	10·68
70 to 80 {Female,..	80	4	61·0	38·37	3·57	2·62	7·63
80 to 90 {Male,.....	99	0	66·7	41·01	4·27	2·83	8·25
80 to 90 {Female,...	79	0	60·0	34·64	3·46	2·37	6·86

According to the experience of Frerichs, the relative weight of the liver in healthy individuals may vary from $\frac{1}{17}$th to $\frac{1}{50}$th of the body; and in adults it fluctuates between $\frac{1}{24}$th and $\frac{1}{40}$th. During the period of greatest growth of the body, the liver does not become enlarged in a manner proportionate to the increase of the entire body; and its diminution in old age is for the most part in advance of that of the body. Its substance in this respect, therefore, presents a marked contrast to the muscular tissue of the heart; for whilst the heart increases progressively up to an advanced age (Bizot, Boyd), the mass of the liver diminishes (Frerichs, Boyd); and in old age, as a general rule, there is senile atrophy of the organ.

Section IV.—Diseases of the Solid Viscera of the Abdomen.

DISEASES CONNECTED WITH THE LIVER AND ITS FUNCTIONS.

Definition.—*Under this heading (as with the previously considered diseases of the abdominal viscera connected with the process of digestion) it is proposed to consider some of the more definite morbid states expressed by functional disorders, lesions of texture, or of both.*

Pathology and Morbid Anatomy.—The morbid states associated with hepatic disorder may be referred—(1.) To congestion of the *liver* in various forms; (2.) To *hepatitis*, or inflammation of its substance, and the formation of (3.) *Hepatic abscess* (*pyæmic, dysenteric,* or *the effect of injury*); (4.) *Chronic enlargement* and *induration;* (5.) To *increased secretion* and *elimination of bile;* (6.) *Jaundice;* (7.) *Acute atrophy;* (8.) *Thickening of the capsule;* (9.) *Cirrhosis;* (10.) *Fatty liver;* (11.) *Amyloid disease of the liver, waxy* or *lardaceous liver;* (12.) *Fibroid deposit;* (13.) *Syphilitic deposit;* (14.) *Cancer;* (15.) *Colloid;* (16.) *Non-malignant tumor;* (17.) *Cysts;* (18.) *Tubercle;* (19.) *Hydatid;* (20.) *Obstruction of vena porta.*

I. **Congestion of the Liver.**—This morbid state is expressed in various forms:—(1.) There may be *congestion from increased secretion of blie,* and the bile-ducts especially are then gorged with bile; or there may be (2.) *Passive congestion* of the hepatic veins, or of the portal veins; and (3.) *Active congestion,* chiefly involving the arterial capillaries.

Morbid anatomy assigns to each of these forms of congestion peculiar and characteristic appearances.

The liver, after death, is found enlarged principally upwards, so as to encroach upon the capacity of the right side of the chest. The ducts are generally distended with bile, and this may arise from various causes, such as compression of the ducts from over-distended veins, frequently brought about by intermittent and remittent fevers. It is also a condition associated with the early stages of *cirrhosis*, when the appearance of the liver is that known by the name of *nutmeg liver*. The distension of the veins seems to be of a passive kind, and to depend on recession of blood from the surface of the body. An ultimate result of hepatic biliary congestion seems to lead to the filling of the hepatic cells with dark yellow matter, and when forms of passive vascular congestion are long associated together, ultimate atrophy and degeneration of the hepatic cells ensue.

The central part of a hepatic lobule is, in health, distinguished by a red spot, centrally situated, and measuring about half the diameter of the lobule. When this dimension increases it is characteristic of *intra*lobular congestion—a morbid state in which the hepatic veins are especially engaged.

In *inter*lobular congestion, where the *portal* veins are specially congested, the centres of the lobules are comparatively pale, while the lobules are surrounded by red vessels.

The condition known as the *nutmeg liver* is explained by the various forms and results of congestion, as well as by other morbid changes of texture. It is so named from the resemblance, in combination and arrangement of colours, in a section of the liver to that seen in the section of a *nutmeg*. In its most marked forms a deep red congestion forms patches and streaks, which occupy the central parts of the lobules, and are *partially* surrounded by patches of a greyish or dirty white colour, and which are devoid of blood. The light-coloured parts may be caused either by fatty degeneration of the cells, which, enlarging, compress the capillaries and expel or prevent their being filled with blood; or it may be due to an increase from deposit and condensation of the Glissonian capsule in the early stage of that morbid condition known as *cirrhosis*. It also is a morbid state in which the capillaries are emptied by compression and contraction of tissue, or in which they have undergone atrophy.

By far the most frequent cause of hepatic congestion is valvular disease of the heart; and especially obstruction to the circulation through the right side, such as results from the causes of *apnœa*. These congestions, so far as the liver is concerned, are of a passive kind; and their most marked and constant effect is to produce *ascites*, with or without *anasarca*. If such congestions continue long, they lead to bilious contamination of the blood.

Congestions of the liver are chiefly brought about by sudden chills, the cold stages of fevers, over-abundant feeding, intemperance in alcoholic or malted liquors: products of faulty digestion are at once carried to the liver, and influence its circulation, and so also does excessive bodily exercise in the heat of the sun. The liver bulges, and may be accompanied by uneasiness on exploration, or of weight on getting into the erect posture. The countenance and complexion may be pale, sallow, or dusky livid; and the tongue will be found coated, the bowels constipated, the appetite defective, and there may be nausea, vomiting, and headache. The pulse is slow, compressed, and irregular, or it may be quick and feeble; and, generally speaking, the symptoms are obscure. Dr. Budd, in his exhaustive work *On Diseases of the Liver*, thus notices how congestion is so common,—"Amid the continual excesses at table of persons in the middle and upper classes of society, an immense variety of noxious matters find their way into the portal blood that should never be present in it, and the mischief which this is calculated to produce is enhanced by indolent or sedentary habits. The consequence often is, that the liver becomes habitually gorged. The same, or even worse effects, result in the lower classes of our larger towns, from their inordinate consumption of gin and porter" (*op cit.*, p. 66).

Treatment.—Congestions of the liver are mainly relieved by a restricted and judicious diet—abstinence from all rich dishes and fermented liquors. Active exercise in the open air must be taken till fatigue is produced sufficient to induce a desire to rest. Horse exercise is especially beneficial.

With regard to medicine, Plummer's pill at bedtime, followed by a seidlitz powder in the morning, or by the usual black draught, or by such saline purgatives as *sulphate of magnesia* and *bitartrate of potash*, which cause a drain from the portal system of veins. An electuary composed of equal parts of *sublimed sulphur* and the powdered gum resin of *guaiacum*, will

be found a most useful remedy in cases where the congestion of the liver is associated with hæmorrhoids, which is extremely common. When dropsy prevails, doses of the diuretic pill, of *hydrargyrum*, *squills*, and *digitalis* (a grain and a half of each ingredient), taken night and morning, are attended with benefit.

II. **Inflammation of the Liver, or Hepatitis,** occurs in various forms, and results in several consequences. In all countries in the temperate zone, at least two forms of inflammation of the liver occur. One of these terminates in simple or granular induration, and has a clinical history of its own; the other terminates in softening and acute atrophy of the gland, associated with jaundice and febrile phenomena of a malignant typhoid type (FRERICHS). In tropical climates a third form of inflammation of the liver has a tendency to result in suppuration; and it is described under the name of *suppurative hepatitis* (ANNESLEY, CAMBAY, HASPEL, BUDD, MOREHEAD, MACLEAN).

The inflammation may have its seat in the fibrous envelope of the gland, or in the sheath of the vessels in Glisson's capsule; or it may attack the glandular parenchyma, the portal or hepatic veins, or the bile-ducts.

Inflammation of the glandular or hepatic parenchyma is either circumscribed, leading to abscess or to granular contraction, or it is diffusely extended over the entire organ; and then, according as the process involves all the anatomical elements of the gland, or is limited to the areolar matrix between the lobules, it gives rise sometimes to softening and acute atrophy, or at other times to induration or cirrhotic degeneration.

III. **Inflammation of the Capsules of the Liver** (*Perihepatitis*) and of Glisson's capsule is rarely accompanied by serious derangements, unless the inflammation extends to the portal or hepatic veins, or causes obstruction of the larger bile-ducts—events of rare occurrence (FRERICHS). Peritonitis, disease of the liver itself, or inflammation of neighbouring structures, such as pleurisy, are the usual causes of *perihepatitis*. The chief symptoms are, tenderness of the hepatic region on pressure, motion, or deep inspiration, without any change in the volume or situation of the organ. Jaundice, as a rule, is absent; so also are febrile phenomena.

IV. **Inflammation of the Glandular or Hepatic Parenchyma** occurs

either as a circumscribed process limited to isolated patches, or it is diffuse, extending over the entire organ in a more or less uniform manner. It is the former variety which leads, in most cases, to suppuration and the formation of abscesses. The diffuse form, on the other hand, at one time induces rapid destruction of the glandular elements, with softening and atrophy of the organ, and at another time it induces induration and cirrhotic degeneration.

In the primary stage of the *acute form of diffuse inflammation of the liver* there occur patches of hyperæmia, while the peripheral portions of the lobules are infiltrated with grey matter, and a juice or fluid serum, rich in albumen, flows from the cut surface of the gland, and the capsule is rough and opaque. Destruction of the glandular cells results in all the cases; but whether atrophy of the gland occurs or not depends upon the stage at which the disease becomes fatal, and partly upon the condition of the liver at the commencement of the process of infiltration. The disease is usually accompanied by a similar condition of the kidneys and spleen, when large quantities of albumen pass off in the urine, and the spleen exists in a state of acute tumefaction.

Violent mental emotions, the condition of pregnancy, typhus, pyæmia, scarlet fever, tropical malarious fevers, are the circumstances under which this form of inflammation is most apt to occur; and the destruction of the liver cell-tissue which ensues is indicated during life by the symptoms of atrophy, and by those peculiar changes in the urine which, at the height of the disease, are almost pathognomonic. In the cases described by Frerichs the urine is described of a brown colour, and smelling of sulphureted hydrogen, having a feebly acid re-action, and containing a small quantity of bile-pigment, but no albumen.

The most important *post-mortem* changes to be seen in such livers are, the occurrence of softened portions, having an irregular form, of a pale yellow or reddish-brown colour. All traces of the outline of the lobules are obliterated, while they are distinct in the firmer portions. The secreting cells of these parts are everywhere destroyed, and their place supplied by numerous oil-globules, granules, and particles of colouring matter. Sometimes the softening follows the ramifications of the portal vein, and the hepatic cells in the softened parts are destroyed, being

converted into a granular debris, oil-globules, and pigment-molecules. In the former portions the entire cells are loaded with fine granules. The gall-bladder, when treated with chloroform, fails to yield pigment. Tyrosine crystals are formed on the cut surface of such livers after some hours' exposure to dry air; and chemical analysis of the parenchyma yields large quantities of leucine and tyrosine. The symptoms of this form of inflammation are included in the account of the disease now known as *acute* or *yellow atrophy of the liver*, which will be given subsequently.

V. **The Chronic Form of Diffuse Inflammation** expresses itself by simple or granular induration of the substance of the liver, sometimes called *cirrhosis, interstitial hepatitis, hob-nailed* or *gin-drinker's liver.* An accurate knowledge of the nature of this induration was first obtained through the investigations of Kiernan, Hallmann, and Carswell. They showed that an increase of the interlobular connecting tissue of the hepatic parenchyma was the essential feature of cirrhotic degeneration. Rokitansky distinguishes two different modes of origin of the granular induration—the one proceeding from a morbid development of the capillary blood-vessels, owing to an excessive secretion of bile; the other due to a chronic inflammation of the hepatic parenchyma, tending to impermeability or obliteration of the finest ramifications of the portal vein. As a rule, the disease only comes under observation when it is more or less completely developed, and when consecutive disorders always associated with it draw attention to the state of the liver.

The commencement of cirrhotic degeneration is marked by increased consistence of the liver. The increased consistence is due to increased and increasing hypertrophy of the areolar framework. The nature of the cell-contents also affects the consistence of the organ. The gland is softer when the cells are loaded with fat, and firmer when they are transformed into *amyloid* (*albuminoid*) material. The amount of blood and of serous infiltration affects the consistence; but the induration of cirrhosis and tenacity of the liver are mainly due to the increased development of the areolar framework.

In the slighter forms the liver may be enlarged or of normal size, rarely smaller than natural; its surface covered by an opaque and thickened capsule, and exhibiting flattened projections, vary-

ing in size from a pin's head to a pea. Similar nodules are observed in section, separated by narrow strips of grey areolar tissue more or less vascular. Sometimes the colour is dark from bile-pigment; sometimes pale from fat.

In the advanced form the liver is reduced in size, particularly the left lobe, which not unfrequently is shrivelled up into a small membranous-like appendage, with a soft flabby rim of connective-tissue at the margins of the organ. Semi-globular knobs, more or less prominent, sometimes of a uniform, at other times of an unequal size and form, are thickly scattered over the surface.

In the lardaceous or amyloid infiltration combined with cirrhosis the liver exceeds its normal size.

The serous capsule is always thickened, and of a greyish-white colour, especially in the depressions between the granulations. Numerous bands of connective-tissue pass from it to neighbouring organs, such as the diaphragm, colon, stomach. On section, the organ has a cartilaginous hardness and coriaceous tenacity, showing at one place narrow, at another broad lines of connective-tissue surrounding the granulations, and sending streak-like processes into their interior. The granulations are generally dark or pale yellow. The elements of tissue are changed as follows:—

(*a.*) *Condition of the Hepatic Cells.*—In such livers a large portion of the hepatic cells are destroyed, and their remains are found in the form of small masses of brownish pigment scattered through the filaments of the newly formed connective-tissue. Another portion of the hepatic cells constitutes the substance of the granulations, and may remain for a long period intact; but ultimately they become filled with fat and various sorts of pigment. In nearly one-half of the cases fatty degenerations occur. The connective-tissue, compressing the commencements of the bile-ducts, causes a retention of the secretion and a jaundiced condition of the liver; hence the deposit of pigment, which accumulates in the form of fine orange or sulphur-yellow granules, more rarely diffused through the cavity of the cells. Another tint may occur from decomposition of the red matter of the blood, especially where branches of the hepatic veins are obliterated.

(*b.*) *The Connective-Tissue.*—Its increased amount presents

numerous varieties as regards its mode of distribution; and to it are due the differences in the size of the granulations.

The earliest increase is usually upon the finer subdivisions of the vessels in the interior of the liver, gradually involving the lobules. The bands of tissue sometimes surround single acini; at another time, three, five, or even seven *acini* may be enclosed. These bands increase at the expense of the *acini*, so that a little mass of brown pigment may be all that remains of the acini; and in this way large masses of connective-tissue take the place of gland-substance. The new connective-tissue varies as to elementary characters: at the circumference of the lobules it is fibrillated; in the substance of the lobules it is disposed to be amorphous; and in the thickened capsule it may be fibro-cartilaginous.

(*c.*) *The Vascular System* of the liver in cirrhosis undergoes great and important changes. The smaller divisions of the portal vein are in most cases narrowed by the shrivelling of the connective-tissue, or by partial impermeability of the finest ramifications of the portal vein from inflammation, obliteration, or compression. The vessels lose their round form, and become angular and bulging. Thrombi ought to be looked for in the branches when destruction of capillaries is obvious; and such destruction of the capillaries of the portal vein is in proportion to the disappearance of the glandular substance of the liver. So long as the hepatic cells exist, the peculiar mesh-like capillary network also exists; but where the cells disappear, their place is supplied by a connective-tissue, and entirely new capillary channels make their appearance, forming elongated meshes, communicating as well with the veins as with the hepatic artery (Frerichs).

The trunk of the hepatic artery also becomes enlarged, and its capillary ramifications more extensive than in the healthy state, black pigment accumulating in its branches. A branched and tortuous network of vessels, of comparatively large calibre, appears in the connective-tissue, their peculiar and tortuous mode of distribution demonstrating their new formation.

The hepatic veins, as a rule, are unchanged; but their obliteration may take place by inflammation, from the capsule of the liver being propagated to the walls of the vessels, when the capillaries of the hepatic vein are gradually destroyed, and their communication with the portal capillaries is interrupted.

(*d.*) *The Bile-Ducts.*—Their origin at the periphery of the lobule is destroyed by the pressure of the new connective-tissue; and there is apt to be catarrhal tumefaction of the mucous membrane of the larger branches.

These various changes may give rise to a long series of functional derangements, which in practice constitute the *symptoms of cirrhosis.* These are mainly—(1.) Derangements of the chylopoiëtic organs, from impediment to the passage of blood through the portal vein into the hepatic veins; and its stagnation in the radicles of the portal vein; (2.) Impairment, passing to complete suspension of the functions of the liver.

Causes.—The chief cause of cirrhotic induration is the abuse of spirituous liquors. In other words, it is due to the specific action of alcohol as an irritant or stimulant poison. Other causes are syphilis and malarious fevers, especially frequent attacks of intermittent fever. The cirrhosis from syphilis is generally associated with the amyloid degeneration about to be described.

Symptoms.—Derangements of the stomach, a loaded tongue, nausea, and occasionally vomiting and faint jaundice, are the earliest symptoms in the clinical history of this disease, which usually comes on very insidiously; and although they may abate, the degenerative process in the liver advances, and gradually undermines the constitution. Digestion continues feeble, and distension and tenderness of the epigastrium, along with heartburn, flatulence, and constipation, are developed. The patients lose flesh and strength, and their colour becomes pale, or dirty yellow, while the skin is dry and rough. The abdomen becomes distended and fluctuates, but the liver is found reduced, and the spleen increased in size; and increasing ascites or tympanitis induces more or less dyspnœa. Hæmorrhages from the stomach or intestines are apt to occur as the disease advances. Enlargement of the superficial veins of the belly may supervene, is characteristic of cirrhosis, and demonstrates that the current of blood in the portal vein is greatly impeded. Slight febrile excitement only manifests itself towards the close, and in most cases diarrhœa terminates life by exhaustion. In other cases the fatal end is by pneumonia, acute pulmonary œdema, or peritonitis. Occasionally death occurs under symptoms of acholia; the patients become jaundiced, purpura spots or ecchymosis are scattered over the skin; while delirium, convulsions, and deep coma close the scene.

Diagnosis.—"Slight sallowness of complexion," writes Dr. Budd, "a dull pain, or some degree of tenderness in the right hypochondrium, with occasional feverishness, in a person above the age of thirty, who has been long in the habit of drinking spirits to excess, are almost conclusive evidence of the existence of cirrhosis, even before there is any direct proof that the circulation through the liver is impeded" (*op. cit.*, p. 158).

Prognosis is always unfavourable; and the main question regarding

Treatment is the possibility of relieving the disorders of function which mainly threaten life, and so to delay the fatal termination. Complete abstinence from spirits is indispensable, and the diet should consist of mild, simple articles of nourishment, especially easily digested animal food. Coffee, spices, and articles irritant to the liver must be avoided. Swelling and tenderness indicate leeches and fomentations. Mild saline laxatives may be given; and when the tenderness ceases, the bowels must be kept open by *rhubarb* and *salines*. When nausea or vomiting occurs, *hydrocyanic acid, belladonna, morphia,* or *extract of nux vomica,* are particularly suitable. When pain prevails, cupping or leeches are indicated over the liver. Saline purgatives, such as *sulphate of magnesia* or *bitartrate of potash,* should also be administered, while iced drinks and low diet must be the rule of life. After ascites is established, bleeding is apt to do much harm; mild diuretics are then indicated, and they are especially beneficial after the operation of "tapping."

VI. **Circumscribed Inflammation and Abscess of the Liver,** or, as it is sometimes called, *suppurative inflammation of the liver,* is always limited to one or to several isolated patches; and, with the exception of congestive turgidness of the contiguous texture, the remaining portions of gland-tissue are rarely implicated. The inflamed patches are usually found in a state of suppuration, which makes its appearance at an early period, gradually destroying the hepatic cells. Sometimes the abscess-cavity is enclosed by a cyst; at other times there is no defined boundary; but the inflammatory process extends till perforation occurs and the pus finds an outlet. The pus is rarely passed into the abdominal cavity; for adhesive inflammation of the capsule covering the abscess almost invariably occurs, so that attachments form to the abdominal walls and adjoining organs. If the

abscess bursts into the abdominal cavity, the result is fatal peritonitis. Frequently the abscess perforates the thoracic or abdominal wall superjacent to the liver, and opens directly outwards; or the pus may discharge itself into the pelvic, inguinal, or sacral regions, close to the spine. Sometimes the abscess tends in an upward direction, to penetrate the diaphragm, when it generally empties itself into the right pleural cavity, but more often forces its way into the substance of an adherent right lung, by a distinctive suppurative process, and in favourable cases passes by a free opening into a bronchus, whence it is discharged. The stomach, the duodenum, and the colon are the principal abdominal organs into which abscesses of the liver discharge themselves.

The hepatic abscesses are sometimes superficial, but more frequently deep-seated, and may be developed in any part of the gland; although they are most frequently found in the posterior portion of the right lobe. In size and in number they vary greatly, although they are rarely more than three, except in cases of pyæmia.

Causes.—Abscess of the liver is rare in temperate climates. It has been caused by—(1.) Contusions; (2.) *Metastatic* or *pyœmic* inflammation, as inflammation of the portal vein, the irritation of veins from disease about the rectum—*e. g.*, fistula in ano, operations for hæmorrhoids; (3.) Inflammation and ulcerative processes in the gastro-intestinal canal, as in dysentery (see vol. i., p. 613); (4.) Inflammation and ulceration of the stomach, gall-bladder, or gall-ducts.

Symptoms.—Fever, rigors, and severe headache and delirium are not uncommon as early phenomena of suppurative inflammation of the liver; but sometimes there are no symptoms pointing to disease of the liver; and the difficulties which embarrass the diagnosis of suppurative hepatitis cannot be over-rated. In 13 *per cent.* the disease runs a perfectly latent course, and in only 8 *per cent.* are symptoms at all well marked (Louis). In most cases a correct diagnosis will only be arrived at by not relying upon individual symptoms; but by taking a general view of the mode of origin and entire clinical history of the case, and after excluding, by comparison, the diseases of the liver and the neighbouring parts which may give rise to symptoms similar to those of hepatitis.

Practically the expression of symptoms denotes either—(1.) A

superficial and adhesive inflammation of the organ; or (2.) A deep-seated suppurative inflammation of the substance of the liver.

The most prominent symptoms of hepatitis are, however, some tumefaction, pain, or uneasiness of the liver, or of the adjoining parts, as the thorax, abdomen, or right shoulder; an affection of the bowels, as diarrhœa or dysentery; and, lastly, pyrexia in a continued, remittent, or intermittent form.

When pain is present, it is found to be in most instances aggravated by lying on the right side, apparently from the greater weight pressing on the liver; while, in a smaller number of instances, the pain is felt most acutely on turning on the left side, probably from adhesions having formed to the ribs. In general, however, the easiest position is on the back, or a little over to the left side; and towards the termination of the disease the patient is sometimes observed lying in a position which he had previously declared himself unable to assume. As the disease advances, the pulse becomes frequent and hard, the skin hot, dry, and constricted, while pain, cough, and dyspnœa increase, and indicate advancing disease (MARTIN). Such a train of symptoms indicates superficial inflammation.

In a few instances acute hepatitis exists without any pyrexia. Some fever, however, is commonly present, and in general it often commences with shivering, vomiting, and purging—symptoms which gradually diminish in a day or two, leaving the patient comparatively free from fever, and the pulse nearly natural. These paroxysms, however, occur at intervals of various duration, sometimes returning as regularly as those of intermittent or of remittent fever; while, in other cases, the periods are less marked, the chief symptoms being rigors occurring at irregular intervals, frequent pulse, and sweats, the latter chiefly occurring in the night, and so copious as in some instances to pour off the body of the patient.

The state of the tongue is usually furred and loaded; but in the course of a long disease it becomes clean, or is only slightly foul. In some few instances, however, it becomes brown and dry.

These general symptoms of suppurative inflammation are insidious in approach, and the destruction of the substance of the liver proceeds silently and rapidly. The existence of the disease is often not known till severe structural changes in the organ

manifest themselves, and then they run a rapid and often fatal course. This is especially the case when the inflammation is induced by the combined effects of heat and malaria. The miasmatic affection of the blood, from deranged mucous surfaces, and from contaminated matters brought by the splenic vein, contributes to hyperæmia of the liver, which is often unnoticed till extensive inflammation develops itself. A burning sensation, with a mottled appearance of the skin of the hands and feet; an irritable temper; a capricious appetite; languor and persistent feverishness; frequent, settled, and increasing pains in the shoulder and back, are all pathognomonic signs of suppurative inflammation going on in the liver. The fever may be from time to time of a continued or intermittent type, and *the patient emaciates slowly*, becoming sad and desponding. The face becomes pale, cachectic, and has a patched or blotchy appearance. Sometimes there is jaundice, followed by anasarca and ascites, intestinal catarrh, bilious diarrhœa, and dysentery. When such signs are observed, it is the duty of a physician to recommend a change of climate (LOWE).

The animal functions, as in phthisis, are often marked by the "cheerful hope" which illumines every hour the patient has to live; but in others the depression amounts to despondency, with restlessness and want of sleep. At last, however, delirium obliterates the past, and throws a veil over the future, till the patient dies.

In the midst of the symptoms that have been mentioned, perhaps an abscess points; and the pus may have formed in the absence of rigors usually indicative of its formation. The patient then becomes hectic, his pulse rapid, and he is covered with a copious and clammy sweat. His life now in a great measure depends on the part where the abscess points; and when it tends to open spontaneously through the thoracic or abdominal wall, the locality is usually the space below the ensiform cartilage of the sternum.

Treatment.—The great experiments which have hitherto been made in the treatment of hepatitis are by bleeding, the use of mercury, and active purgatives; and it may be affirmed as a general result that blood-letting will not cut short the morbid process in acute hepatitis; and Frerichs is of opinion that it should only be ventured on in cases of traumatic hepatitis, and in robust

plethoric persons, where there is great tenderness, with enlargement of the liver, and urgent dyspnœa. In the young and sthenic European in the East Indies, it is in general necessary to take fifteen to twenty ounces of blood, or till the skin becomes soft and relaxed, or the pain abates. Much benefit may be derived from the local application of leeches and large blisters.

One practical rule seems to be established with respect to the use of mercury in the treatment of hepatitis, which is, that it is not only inefficient, but injurious, and should not be given except as an occasional purgative. When congestion is known to exist, leeches may be applied with benefit, combined with purgative extracts and antimonials. The local abstraction of blood is also efficiently accomplished by leeches round the anus. They act more directly on the portal circulation than over the hepatic region. Ice applied to the region of the liver is also of great service, and the diet should be as limited as is consistent simply with the maintenance of life.

Purgatives are of use when the intestinal functions are sluggish.

In considering the treatment of abscess, and especially the points bearing on surgical interference, it should be remembered that hepatic abscesses tend to point or empty themselves in the following directions. These are stated in the order of their relative frequency:—(1.) Through the lungs (from 10 to 30 *per cent.*); (2.) Through into the peritoneum, the stomach, or some part of the intestinal tract (nearly the same proportion); (3.) Through the external integuments (in the minority of cases).

With regard to the treatment of abscess when it tends to make its way outwardly, it is still a question whether or not an artificial opening ought to be made, the prominences of the false ribs and obliteration of the intercostal spaces being considered sufficient, in the absence of fluctuation, to justify the operation (Frerichs). In its performance care must be taken to prevent the entrance of pus into the abdominal cavity. With this object in view, the following method has been recommended by Bégin and Recamier:—(1.) Being decided as to the limits of the abscess, the patient is (2.) laid on his back, with the upper part of the body bent forward and the thighs flexed upon the abdomen; (3.) An incision from two to three inches long is to be made over the abscess, dividing the skin, the subcutaneous adipose tissue, the muscles, and the aponeurosis; (4.) The peritoneum is then

laid open, as in operating for hernia, by slitting it up on a grooved director to the same extent as the primary incision; (5.) The wound is then to be dressed with charpie, and to remain untouched for three days; (6.) At the end of three days the dressing is to be removed, when the capsule of the liver will be found to have contracted adhesions to the margins of the wound so firm that the abscess may be opened without the danger of pus passing into the abdominal cavity.

Dr. Budd mentions the following expedient as a method of indicating whether the liver adheres to the abdominal parietes or not. When the liver is large, and the abdominal parietes are thin, it may be made out "by feeling the edge of the liver, or some prominent part of its surface, and marking the place of this with a pen on the surface of the belly. If the liver be adherent to the abdominal parietes, the line or spot so marked will correspond to the edge or prominence of the liver in all positions of the body. If it be not adherent, the liver will slide along the wall of the belly when the patient draws a deep breath, or changes his posture—the liver will fall, for example, towards the left side when he turns from his back over to that side, and the line or spot will no longer correspond to the edge or prominence in question" (*op. cit.*, p. 122).

A simple puncture is only warrantable when the pus has already penetrated through the superficial layer of the abdominal aponeurosis, or the intercostal muscles. In all other cases it ought to be avoided as dangerous. Indeed, Mr. Lowe (whose valuable records of cases of hepatic abscess in the *Madras Quarterly Journal* for April, 1863, merit the most careful study) thinks it better to allow an abscess of the liver pointing through the abdominal wall to open of itself, for the following reasons:—(1.) Because of the inelastic structure of the lobular substance of the liver not permitting the cavity to contract when a free opening has evacuated the pus; (2.) Because air invariably enters when an artificial opening is made, and rapid decomposition of the pus takes place, and renewed inflammation of the walls of the sac sets in; (3.) This renewal of inflammation and fever may end in gangrene, and may thus rapidly prove fatal; (4.) When the operation is entirely left to nature, *small worm-eaten-like openings serve to discharge the pus*, so that it has slow but constant escape. As these apertures never close up, and as the matter is

always oozing out, air cannot enter, no decomposition takes place (no septicæmia occurs), and no secondary fever sets in. The patient feels no shock from the loss of the matter, which escapes so gradually, and as it escapes, so Nature closes up the walls of the sac. Such is also the opinion and advice of Dr. Budd.

But others consider that the abscess ought to be evacuated under certain conditions. For example, a most interesting case of hepatic abscess is related by Sir Henry Cooper, of Hull, in the *British Medical Journal* for May 23, 1863. Sir Henry Cooper justly takes exception to the method of Bégin and Recamier, because it may provoke the very danger it is desirable to avoid; and he believes that in all cases in which the suppuration has extended so near the surface of the liver as to give the sense of fluctuation, irritation and adhesion of contiguous surfaces have taken place. The process also interposes a serious delay at a critical period. Therefore he advocates a direct opening into the abscess, *when there is reasonable ground for believing that an external outlet for the matter is the direction taken by the abscess.* Peritoneal connections are sure then to have taken place. Tenderness of the tumor he regards as the most satisfactory indication for the operation, and a hardened base of effused lymph a certain confirmation. Delay exposes the patient to the risk of rupture of the walls of the abscess by coughing, sneezing, or the like, and to the laceration of any adhesions which may have formed; or it allows him to perish unrelieved from the effects of constitutional irritation and consequent exhaustion.

A middle course was devised and carried out by Dr. Graves, in the Meath Hospital, in the case of a robust man. External elevation, hardness, and pain, eventually confined to one spot in the right hypochondrium, after acute inflammation of the liver, left no doubt of the formation of an abscess. The hardness was followed by deep-seated softness, yet no tendency was shown by the abscess to point outwards. The swelling remained stationary, and the integument of natural colour; but the general strength began to fail, and it became an important question whether the abscess should be opened by operation. It was objected—(1.) That the external tumor was diffuse; (2.) That the exact site of the abscess was uncertain; (3.) That failure to evacuate its contents might prove detrimental. A decision was therefore given *against* operation, by the surgeons of the Meath Hospital. Under these circum-

stances, Dr. Graves remembered that he had seen several cases where an incision made over a deep-seated abscess had failed to give vent to the matter in the first instance; yet in the course of a few days the pus found its way to the incision, and burst through it. He therefore proposed that an incision about four inches long should be made, exactly over the centre of the tumor—that it should be carried through a considerable depth of muscle, and, if possible, be continued *to within about one or two lines of the peritoneum.* This incision was then to be plugged up from the bottom of the wound with lint, and thus kept open, in the hope that the pus might tend towards the incision, and finally burst through it. Such an operation was performed. The abdominal muscles were found of considerable thickness, and quite healthy; and although the incision was very deep, yet the situation of the hepatic abscess was not felt more distinctly, so that it now became quite evident that no prudent surgeon would have persevered in an attempt to open directly into it. A result was therefore waited for. In two days afterwards the patient sneezed, when purulent matter in very large quantity burst forth through the wound. On closer examination it appeared that the incision had not been made exactly over the abscess, but rather to one side of it; for the matter did not come from the bottom, but from the side of the wound, and pressure on the liver on that side caused matter to flow out in abundance through the wound, which communicated laterally with the abscess.

This case teaches that if the attempt had been made to open the abscess directly, by continuing the incision, it would have failed, and the opening into the peritoneum and liver perhaps proved fatal (*Dublin Hosp. Report,* vol. iv., p. 40).

After the abscess has opened, strict rest must be enjoined; and sometimes the cavity is very long in closing up. Convalescence is always tedious; and sometimes the cicatrization of the abscess is imperfect, continuing to discharge pus at intervals for years (Frerichs).

Because the liver has been repeatedly punctured (it is said, deeply) with an ordinary trocar without any evil consequences, beyond slight local irritation, it has even been argued that our efforts should be directed to detecting by acupuncture the seat of an abscess, and evacuating it as soon as possible; and that this method of exploration or "prospecting the liver" should

be commenced as soon as symptoms present themselves indicating abscess of that organ! It has been argued that because some cases have recovered after such haphazard methods of finding and evacuating an abscess, it is a practice which should be made the rule, as already stated, in place of waiting for some indications that pus has even formed in the liver (MURRAY, CAMERON). Statistical data regarding the actual value, or even the safety, of this method of thrusting trocars into the liver in search of abscesses have not been published; therefore the recommendations to the plan rest upon the opinions or impressions of two or three individual men, the records of whose practice are unknown. Certain it is that the plan recommended is opposed to the well-established principles upon which surgical interference rests, and is opposed to all that is known regarding the pathology of hepatic abscess, as set forth in the carefully recorded facts of Waring, Martin, Morehead, Frerichs, Budd, Maclean, and Lowe.

It is also stated by Dr. Cameron (the latest exponent of this method of puncture), that in cases of hepatic enlargement, where the trocar has been used more than once in an unsuccessful search for an abscess, the operation was followed by the speedy absorption of an enlargement of the liver, which had resisted all the routine methods of bringing about its dispersion—and that this is a native mode of dispersing hepatic and even splenic enlargements in India (*Lancet*, August 8, 1863, p. 169).

Puncturing parts, or acupuncture with a very fine needle, is a surgical operation said to be much in use amongst the native Chinese and Japanese hakims; but the statement requires confirmation, and accurate information regarding the results of such operations are not on record. It is obvious, therefore, that such modes of dealing with large livers, or *probable* hepatic abscesses, cannot be recommended here; and it should be remembered that a hepatic abscess always begins from multiple foci (ROKITANSKY). But we must still look to physicians of large Indian experience for information on this important subject.

The time which elapses after opening an abscess till the patient's recovery is generally from one to two months. When convalescence is established, the functions of the liver remain torpid and its substance often indurated; and subsequently, also, the spleen may be enlarged. In such a state Sir Ranald Martin recommends the use of *nitro-muriatic acid* baths. They promote

the depurative functions of the liver, kidneys, bowels, and skin.

"The form and manner of preparing and using the acid bath are as follow :—Take of hydrochloric acid three parts; nitric acid, two parts; mix the two acids carefully and slowly, so as to avoid evolution of heat; and having waited for twenty minutes, add of distilled water five parts, and mix the whole carefully.

"*For a General Bath in which to Immerse the whole Body.*—(1.) Pour into the bath about five pailfuls of cold water; add two quart bottles, containing sixty-four fluid ounces of the dilute nitro-muriatic acid, prepared as above, and then sufficient boiling water to raise the temperature to 96° or 98°. The body is to be quickly and thoroughly dried with warm towels; and afterwards the patient must retire to a well-aired and warm bed. The use of the bath is only to be discontinued when tenderness of the gums or general *malaise* occur; and cuticular irritation from the acid is to be avoided by diminishing its strength. Iron and other forms of tonic remedies may also be administered at the same time, as well as opiates," (Martin *On Climate*, p. 564, *et seq.*)

VII. **Increased Secretion and Elimination of Bile.**—This condition is brought about by some increased excitement of the liver, as by certain kinds of food or drink; but more especially, as is now well known, it is brought about in Europeans by exposure to unusually high ranges of temperature. In summer and autumn it is a morbid state not uncommon in our own country; but to the European on first landing in India it most commonly occurs, and in either case it occasions what is termed a "bilious diarrhœa." Great increase of temperature, combined also, perhaps, with change of climate and an unsuitable mode of life, is now acknowledged to have a direct influence on the functions of the liver, expressed, in the first instance, by an unmistakable increase in its secretion. As a complication of other diseases, hepatic disorders are of most frequent occurrence, and for various reasons no true estimate has yet been made relative to the frequency of hepatic disease either in this or in other countries. Like most other exciting agents, however, the prolonged exposure to the influence of increased temperature, under conditions such as are experienced by the European in India, ceases to have a stimulant effect, and a depression in the powers of the organ results, corresponding to the previous excitement. In India the duration of the exaltation of hepatic function is not found to be of long duration, but is

confined to the earlier years of residence, declining from that time. This increase of secretion and its elimination, so long as it lasts, is justly regarded as a salutary effort which nature provides to maintain the health. It is the decline of the powers of secretion, the arrest of the function, and the more or less sudden suppression of the secretion which is attended with danger. It always precedes the expression of the inflammatory tendency, and is associated with congestions of an active kind, in which the arterial system becomes prominently excited. This is consistent with what occurs in every other secreting organ. Previous to the expression of the inflammatory process becoming developed in a gland, the secretion at first flows freely under excitement (not necessarily morbid); but so soon as the inflammatory tendency becomes expressed, the secretion is at the same time dried up. This is well seen in the phenomena of a common cold, when the exciting cause at first merely stimulates the nasal passages, as marked, for instance, by an increased flow of mucus from the nose; but so soon as the inflammatory state becomes fully expressed, the secretion is dried up, and when re-established, is unmistakably altered in its properties. So it is with the liver when the increased flow of bile is suspended, from exposure to cold or some other exciting cause, a warning is given that the inflammatory tendency is about to be expressed by congestive and exudative processes in the texture of the organ.

Symptoms.—With excessive secretion of bile, the patient has purging of bilious stools, causing scalding of the anus. There is slight sickness just before the bowels act. A bitter taste is felt in the mouth, and the tongue is foul.

Copious draughts of hot water act as an emetic—dilute the bile; and if some saline purgative be added, the congestion of the liver will be relieved. If pain prevails over the liver, leeches and fomentations are indicated, to be followed by Plummer's pill at night and a black draught in the morning. The diet must be extremely limited and strictly regulated, as already noticed under congestions of the liver.

In persons who die soon after arriving in India, Mr. Twining remarks that the gall-bladder is commonly distended with bile.

VIII. **The Degenerations of the Substance of the Liver** consist of —(1.) *The amyloid;* (2.) *The fatty;* and (3.) *The pigmentary;*

the general pathology of which have been already fully considered in the first volume of this text-book, p. 120.

1. *The Amyloid Degeneration,* though frequently associated with fatty degeneration, has no necessary connection with it, but is an immediate alteration or degeneration of the glandular hepatic cells (BENNETT, FRERICHS).

The amyloid liver is usually pale or fawn-coloured—sometimes congested, so that it has a distinctly mottled appearance. The limits of the acini are unusually well defined, and the section or cut surface appears smooth, dry, and firm. The tissue is extremely slow to decompose ; its specific gravity is notably increased, in some instances above 1080 ; but it may be reduced by fatty degeneration co-existing. Microscopically the entire structure seems changed, but it does not appear in which tissue the change begins. Meckel says it begins probably in the cells, and Virchow has traced its origin very clearly here. The peculiar structural formation of the liver has enabled the advance of the degeneration to be observed. His observations show that,—First the branches of the hepatic artery (intralobular vessels) are affected ; and when the degeneration has become established in an acinus, three zones, varying in colour and appearance, are observable,—(1.) The outermost is composed chiefly of portal vessels, and is generally the seat of more or less fatty degeneration; (2.) The innermost zone is made up of the intralobular vessels (of which the vein is prominent in the centre when divided in section); these are surrounded by pigmentary deposit normal to the liver; (3.) The intermediate zone is the site of the amyloid material, translucent and firm. After the vessels have degenerated, the secreting cells about them become involved. They acquire a homogeneous hyaline appearance, like particles of rough ice. They lose their nuclei. A cell-wall cannot be seen ; but they appear as an ill-defined, pellucid, glistening mass.

When the degeneration exists in an extreme degree, the greater is the translucency of the tissue, and the pellucid appearance gradually extends over the entire area of a lobule, and the few blood-vessels it seems to possess emit a little watery blood only. Rarely it occurs in isolated patches ; generally it is distributed throughout, but more marked in some places. Towards the circumference of the liver, and shining through the clear peritoneal covering, the lobules or acini may be seen mapped out in a

remarkably defined manner; in fact, in no disease of the liver is the appearance of lobules (which as a rule are not marked) so distinct as in this particular morbid condition (Wilks's *Guy's Hospital Report*, p. 123, 1856). This is due to the way in which the change begins and progresses. The fatty degeneration at the margins makes the acini still more clearly obvious; and the approximation of the two degenerations (fatty and amyloid) gives an opaque white appearance, which encircles the acini and maps them out in this perfectly defined manner. A section well washed will also show the dead-white opaque substance running in the course of the portal vessels, surrounding the more transparent substance of the acini, in the centre of which the hepatic vein is apparent.

By these characters the lesion must be distinguished from the simply fatty degeneration of the liver; for it is now certain that up till the year 1854 these two forms of degeneration have often been confounded. But in the appearances shown by the livers affected by these several degenerations the differences are well marked. Louis evidently confounds the waxy and fatty liver in phthisis under the latter description; and the confusion has been transmitted from this great authority.

The large and hypertrophic livers, in their extreme degree, are for the most part due to the waxy degeneration, either simple or combined with the fatty degeneration; whereas the fatty degeneration is often present in the liver to an extreme degree without much or any hypertrophy. In the purely fatty liver, also, the specific gravity is not raised, but reduced. It is recorded as low as 1005. When the fatty co-exists with the waxy degeneration, the specific gravity of the organ may be normal. Fatty liver consists of precisely the same elements as the normal liver in a similar structural arrangement, but an essential element is morbidly increased in its cells. The aggregate of solids and water may be slightly increased or diminished, but the *per centage* on the whole is reduced. Oil may be deposited to an enormous extent in the gland-cells; and these growing or swelling out, the volume of the organ, as well as its weight, may be increased to a very great extent.

A form of cirrhosis is due to the waxy liver, as well as to the fatty. In the former it assumes at once the *atrophic* form; and all the essential elements simultaneously disappear. The atrophy

commences in the glandular epithelium or hepatic cells, and thus gives rise to an apparently relative hypertrophy of the fibrous tissue.

On the other hand, cirrhosis is a complex process, a lesion due to actual growth, hypertrophy, and contraction of connective-tissue.

Characters of the Minute Tissue-Elements in Amyloid Liver—(1.) *Gland-Cells.*—The finely granular contents gradually disappear, giving place to a homogeneous, clear substance, which ultimately replaces the cell. In a few hepatic cells the mark or trace of the nucleus may still be seen of a shining lustre; and, when completely altered, the cell resembles a brilliant pellucid homogeneous scale. The cells, so degenerate, are excessively coherent; not easy to separate; and, indeed, so changed that a large solid aggregate mass is alone recognizable, in which neither cell, nor areolar matrix, nor blood-vessel is to be distinguished.

(2.) *Blood-Vessels.*—The walls of the more delicate vessels become thickened, rigid, homogeneous, and lustrous. Their channel is narrowed, and ultimately entirely obliterated; while the still patent portions remain patulous on section. They appear as pellucid, transparently colourless cylinders, in which no trace of the delicate structure of blood-vessels can be detected.

It is difficult to determine to which system the vessels so affected belong; but, so far, it is an ascertained fact that the minute ramifications of the hepatic artery are chiefly implicated in the first instance. Frerichs has "repeatedly observed diseased capillaries, the locality of which appeared to correspond to the situation of the portal and hepatic veins. Solution of iodine gives a deep red colour. The careful and slow addition of sulphuric acid changes the deep red to a dirty violet, and rarely to a blue tint. The tissue which remains exempt from the degeneration is distinguished by the greater amount of blood, and by greater softness and moisture. The chemical character of the amyloid liver has been already considered in the first volume; and it may simply be noticed here that Frerichs has especially examined into any relations which may subsist between the *glycogenic material* normally elaborated by the liver, and the *amyloid substance* which is the result of the morbid process now under consideration. Both substances are *coloured* by iodine; but a chemical re-action takes place in the

case of the *amyloid substance,* and both are regarded as carbo-hydrogens. The results are mainly negative—sugar being furnished by the *glycogenic material,* when digested with saliva, or when brought in contact with albumen, but not by the *amyloid substance.*

This form of disease has long been known as a liver affection; and Rokitansky first gave a clear account of its essential characters, and recognized the relation of the lesion to certain *cachexia.* Budd describes the affection as a "scrofulous enlargement," and Oppolzer as the "colloid liver."

Diagnosis.—Frerichs relates a case of waxy liver in a girl aged nine years, in whom ascites was present; and the fluid drawn off contained a large quantity of sugar. With the exception of cases of diabetes, sugar has never yet been found in ascitic fluid; and therefore its presence, it is suggested, may be diagnostic of waxy liver. Dr. Wilks found a trace of sugar in the fluid from the peritoneum of a similar case. There are also general and local grounds for suspecting the existence of amyloid disease. The several symptoms may be stated as follow:—

1. When there is general ill-health, expressed by *marasmus, anæmia,* or *dropsy,* which constitute the primary symptoms in cases otherwise ambiguous, and which in some cases are associated with diarrhœa, vomiting, and cardiac systolic murmur.

2. In cases where (after examining the blood) such symptoms as are mentioned cannot be traced to lesions of such organs as we have hitherto been accustomed to refer these phenomena.

3. In cases where the constitution is enfeebled, and health is impaired by *ulcerations of bones, syphilis, intermittent fever, tuberculosis, malaria.*

The local indications of amyloid liver may be thus given from Frerichs's cases:—

(1.) Uniform enlargement of the organ; (2.) Increased consistence, indicated by firmness; (3.) Association of these characters with tumid spleen and albuminous urine; (4.) Association of these characters with any of the conditions enumerated in the general symptoms under (3).

The prominent general symptoms of this fatal degeneration being "anæmia, prostration, exhaustion," the condition of the liver, spleen, and kidneys should be investigated in all such cases, and their condition recorded in all cases of *syphilis, caries,*

necrosis, and *intermittent fever;* and it is desirable to limit the suppuration of bones as much as possible by early surgical interference. Death usually occurs by exhaustion, unless a rapid end is brought about by *purulent peritonitis, dysentery, pneumonia, gangrene*, or *œdema of the lungs.*

2. *Fatty Degeneration of the Liver* is due to an abnormal increase of the fat naturally contained in the hepatic cells, so that they become engorged with oil. The nuclei are thus obliterated or obscured. The morbid condition is generally associated with free living, the influence of *alcohol*, of *syphilis*, the deprivation of exercise, and exposure to a heated air, with a large supply of food.

The Symptoms are chiefly gastric catarrh, indigestion, diarrhœa, anæmia.

3. *Pigmentary Degeneration of the Liver* is only to be recognized at a *post-mortem* examination. It is seen in cases of severe remittent, intermittent, or malarious fevers. It is due to the accumulation of pigment-matter in the vascular apparatus of the gland, especially in the capillary network of the portal and hepatic veins; and the minute branches of the hepatic artery also contain quantities of black colouring matter. In cases where the liver is so affected, similar melanic matter is generally found in the spleen, kidneys, and brain; while the blood itself may be seen to contain dark granular masses, or nucleated pigment-cells, with black granules in their interior. The spleen seems to be the seat of formation of the melanotic matter (FRERICHS), whence it passes into the portal vein.

The effects upon the system of this degeneration are mainly due to the destruction of blood-corpuscles with which it is associated, and tending to a condition like that of *chlorosis.* The bile is usually secreted in large quantity. There is extensive capillary stagnation, which gives rise to obstruction of the circulation of the blood in the roots of the portal veins—and exhausting hæmorrhages of an intermittent kind are apt to occur from the gastro-intestinal mucous membrane. Profuse diarrhœa, vomiting, and serous effusions are also common occurrences. In India it tends to *suppurative hepatitis.*

Treatment of Degenerations.—The main indications consist in the removal of the causes, or the cure of the disease which has induced the degeneration. Easily digested and nutritious food

must be given, and especially where chronic congestions prevail, a strict diet plays a powerful part in effecting a cure. Alcoholic drinks, sugar, and fat are to be avoided, as well as amylaceous substances. *Aloes, rhubarb,* or *sulphate of soda,* are useful to remove the torpidity of the bowels. *Muriate of ammonia* is recommended by Dr. Budd, in doses of from *five* to *ten* grains three times a day. He believes it probably relieves the liver, and does good by promoting the action of the skin and the kidneys.

IX. **Jaundice, Icterus,** or the **Yellows,** are terms which comprehend a group of diseases, many of the different tissues and fluids of the body being dyed yellow, but more especially the conjunctiva and the connective-tissue.

Pathology.—Jaundice, though often a result of every organic disease of the liver or duodenum, yet often occurs when those organs are perfectly healthy or simply congested. On posthumous examination, besides the yellowness of the cutis, the serum of the blood is generally found loaded with bile, and perfectly yellow. If the disease has continued for some time, the fat is also yellow, as well as the bones and cartilages, the serous fluids, and even the milk expressed from the breast of the female.

The theories that have been advanced to account for jaundice are, that the bile exists formed in the blood, and is merely removed by the liver, and that consequently jaundice is a consequence of the non-separation of the bile. A more common opinion is, that bile is a secretion, and not a mere separation, and consequently that in jaundice the bile is first secreted and then absorbed both by the veins and lymphatics; while Portal has proved that it may be absorbed also by the lacteals. The theory of jaundice especially advocated and expounded by Dr. Budd is, that the disease may arise in two ways,—(1.) By mechanical obstruction to the passage of the bile into the intestines, and the consequent re-absorption of the detained fluid into the blood; (2.) The suppression of the biliary secretion arising from some morbid condition of the liver itself, whereby biliary ingredients accumulate in the circulation. Some of these ingredients or constituents of bile are generated in the liver itself (*e. g.,* the bile acids); others exist pre-formed in the blood (*e. g.,* the green bile-pigment, or *biliverdine,* and the *cholesterine*).

The mechanism of jaundice has therefore been regarded from

two points of view, namely,—(1.) Jaundice from suppression, retention, or non-elimination (BUDD, HARLEY); (2.) Jaundice from re-absorption of bile (FRERICHS).

The former of these forms of jaundice is characterized by the rapid accumulation of green bile-pigment in the blood, until the serum, and the tissues, and the urine are saturated with the pigment. Over this secretion the mental state seems to exercise a very remarkable influence—so much so that mental emotion, favouring congestion of the organ, favours the stoppage of the secretion. Thus jaundice by suppression or non-elimination arises from (1.) Innervation (HARLEY). Active and passive congestion similarly induces jaundice; hence jaundice by suppression or non-elimination arises also from (2.) Disordered hepatic circulation (HARLEY). (3.) Another form of jaundice from suppression or non-elimination arises from loss or destruction of the secreting cells of the liver, as in acute and chronic atrophy, cancer, tubercle, fatty and amyloid degeneration. Hence jaundice by suppression or non-elimination arises also from the loss or absence of the glandular hepatic substance (HARLEY).

The second class of cases of jaundice arises from the re-absorption of the secreted but retained bile. They are also characterized by the accumulation of pigment in the blood; whence it stains the tissues, the urine, and the serum. The bile in these cases is absorbed from the distended ducts and gall-bladder; and the biliary products manufactured in the liver, equally with those formed in the blood, find their way back into the circulation, to be eliminated by the excretions. Hence the bile-acids (absent in the former class of cases of jaundice) are present in cases of jaundice from re-absorption, as well as the bile-pigment. Obstruction may be due to several causes, but chiefly to the two following, namely:—(1.) Congenital deficiency (very rare); (2.) Obstruction by disease, generally of parts in the vicinity of the head, of the pancreas, or of the *ductus communis choledochus*.

Symptoms.—Jaundice, from the different intensities of the colour of the skin, has been divided into the *yellow*, the *green*, and the *black* jaundice, arising from functional disease. Jaundice may be sudden in its attack, or it may be preceded for a few days by great depression of spirits, lassitude, and somnolescence. It may also be preceded or accompanied by some slight pain in the region of the liver, but more commonly pain is not present.

The first symptom of jaundice is a yellowness of the white of the eyes, then of the roots of the nails. The yellowness next appears over the face and neck, and ultimately over the trunk and upper and lower extremities. As soon as the eyes are affected, the urine becomes of a deep red colour, and stains linen yellow, and if nitric acid be added, it is changed to a deep green. The bile, however, is not always in the same state of combination in the urine, nor of the same quality; for in some instances, where the colour of the patient is most marked, and the urine of its deepest hue, the addition of nitric acid effects no change. At the same time that the urine is thus discoloured, the stools, often abundant in quantity, are copious and white. The pulse is slow, and the patient complains of a bitter taste in the mouth, has much thirst, an absolute inaptitude for all exertion, and suffers from a lowness of spirits amounting to hypochondriasis. In general the bowels are irritable and easily acted upon; but in a few cases they are constipated. If the patient recovers, the first symptom of recovery is the appearance of bile in the stools, after which the yellowness fades away in the inverse order of the attack.

The duration of this affection is such that in some cases it terminates in about ten days, but more generally it lasts from three to six weeks, and, if badly treated, oftentimes as many months.

Diagnosis.—This disease is to be distinguished from *chlorosis* and that sallow state which results from profuse uterine hæmorrhage. In these complaints the white of the eye is clear, the urine limpid, and the stools healthy, so that the great characteristics of jaundice are wanting.

The difficulty is to determine how far the jaundice is due either to obstruction or to non-elimination (suppressed secretion); and to Dr. Harley we are mainly indebted for methods of clinically distinguishing these two classes of cases by chemical examination of the excretions. His principle of diagnosis from this point of view assumes that in jaundice from suppression of bile, only those biliary products which exist pre-formed in the blood accumulate in the circulation; but that in jaundice from obstruction, the biliary products which are manufactured in the liver, equally with those pre-formed in the blood, find their way back to the blood, to be eliminated by the excretions.

The absence of bile from the stools is indicated by their pipe-clay colour, offensive odour, and presence of fat.

The urine of jaundice ranges from a saffron-yellow to a dark olive-green, or even black colour; and its amount of pigment is best judged of by the intensity of the colour of the uric acid crystals. But it is the presence of *biliary acids* in the urine which Dr. Harley considers characteristic of jaundice from re-absorption, as distinguished from jaundice arising from non-elimination or suppression.

The readiest mode by which the biliary acids may be detected is the following:—

"To a couple of drachms of the suspected urine add a small fragment of loaf-sugar, and afterwards pour slowly into the test-tube about a drachm of strong sulphuric acid. This should be done so as not to mix the two liquids. If biliary acids be present, there will be observed at the line of contact of the acid and urine—after standing for a few minutes—a deep purple hue. This result may be taken as a sure indication that the jaundice is due to obstructed bile-ducts. On the other hand, the absence of this phenomenon, and the occurrence of merely a *brown* instead of a *purple* tint, although in the earlier stages of jaundice equally indicative of suppression, is no indication of the cause of the suppression, which must be gleaned from other circumstances" (Harley *On Jaundice*, p. 61).

Dr. Felix Hoppe's method is as follows:—

"(1.) Decompose the icteric urine to be examined with an excess of milk of lime; (2.) Boil for about half an hour; (3.) Filter; (4.) Evaporate the filtered fluid nearly to dryness; (5.) Decompose with a great excess of concentrated hydrochloric acid, and then keep the whole (before being again filtered) at the boiling-point for half an hour; (6.) To avoid spurting of the fluid, it is necessary to renew the volatilized hydrochloric acid from time to time; (7.) Leave the liquid to get completely cold, and then add six to eight times its volume of water; (8.) Filter the dark-brown turbid solution thus obtained, wash out with water the residue on the filter, until the same runs through quite colourless; (9.) Dissolve the brown resinous mass on the filter in 90 *per cent.* alcohol; (10.) Decolorize by boiling with animal charcoal, filter, and evaporate to dryness in the water-bath; the residue is a yellow, resinous mass, which, if bile-acids be present, must consist, for the most part, of pure *choloidic acid.* In such a case it melts by warming, and emits the peculiar musk or soap odour. (11.) Lastly, dissolve in a very little caustic soda and some drops

of warm water, add a very small piece of sugar, and allow three drops of concentrated SO_3 slowly to fall into it. At first the fluid becomes milky and troubled, and resinous flakes separate, which stick pertinaciously to the glass, but afterwards, by the addition of more SO_3, these again dissolve, and produce a beautiful purple-red or dark-violet fluid." ("Abstract of Kühne's Paper on the Pathology of *Icterus*," by Dr. George Scott, of Southampton, in Beale's *Archives*, vol. i., p. 343).

But these acids after a time gradually diminish and disappear from the urine in cases of obstruction and re-absorption—namely, when the secreting powers of the liver become impaired, when the hepatic gland-cells waste away; and then the abnormal products *leucine* and *tyrosine* appear in the urine. They are readily detected by the slow evaporation of an ounce of urine to the consistence of syrup, which is to be put aside to crystallize. *Tyrosine* is recognized by the fine stellate groups of needle-like crystals or spiculated cells, which appear like a rolled-up hedgehog with its bristles sticking out in all directions (HARLEY). It may be obtained in its pure state by (1.) Adding a solution of *acetate of lead* to four ounces of urine till a precipitate ceases to form; (2.) Filtering; (3.) Freeing the liquid from an excess of lead by a current of sulphureted hydrogen; (4.) Again filtering; and (5.) Evaporating the clear solution. The tyrosine is now colourless, and crystallizes with the microscopic characters better marked. *Leucine* is known by its flat, circular, oily-like discs, without crystalline structure. It is soluble in water and boiling alcohol, but insoluble in ether.

If sugar appears in the urine in cases of jaundice, with a diminution of bile-acids hitherto in quantity, it is generally the forerunner of a fatal termination (HARLEY).

Treatment.—As a general principle, the larger number of cases of jaundice from functional disorder (perhaps four out of five) will get well in time spontaneously, but may be aided by remedies judiciously selected according to the diagnosis already indicated. The first indication is to aim at removing the exciting cause; and in jaundice due to congestion of the liver, purgatives seem to act beneficially in the form of *blue pill* or *Plummer's pill*, with *aloes*, and *nux vomica* with *rhubarb pill mass*. Acids and alkalies are alike counter-indicated in cases of jaundice resulting from active congestion of the liver. *Benzoin acid* has been recommended; and Dr. Harley has found it useful in cases of

suppression of bile (by innervation, for example); but in cases of jaundice from obstruction it is injurious. *Podophyllin* is also of use in jaundice from suppressed secretion of bile. It ought to be combined with *hyoscyamus*. It is especially useful in cases of feeble liver, combined with vegetable tonics, such as *gentian* and *quinine;* but it ought not to be given in cases of jaundice from obstruction. The medicine of all others which has seemed most generally useful in Dr. Budd's experience is *sulphate of magnesia*, in half drachm to drachm doses, combined with fifteen grains of *carbonate of magnesia*, and half a drachm of *aromatic spirits of ammonia*, given three times a day an hour before food. The *sulphate of magnesia* maintains a free action of the bowels; and the *carbonate of magnesia* neutralizes any excess of acid in the stomach or bowels (*op. cit.*, p. 289).

ACUTE OR YELLOW ATROPHY OF THE LIVER.

LATIN EQ., *Atrophia acuta;* FRENCH EQ., *Atrophie aiguë;* GERMAN EQ., *Acute Atrophie;* ITALIAN EQ., *Atrofia acuta.*

Definition.—*Simple jaundice followed by violent constitutional disturbance, expressed by pyrexia, delirium, hæmorrhages from various parts, and finally coma. The liver shrinks to one-half or one-third its normal size. A peculiar chemical decomposition takes place in the liver, whereby abnormal proximate principles are formed, which, being carried into the blood, may be discovered in various organs of the body, or, passing out by the kidneys, may be found in the urine.*

Pathology and Symptoms.—The disease was first described in this country by Bright and Graves, and more recently and minutely by Frerichs and Wilks. The liver undergoes a most remarkable atrophy, and its section presents a mottled red and yellow appearance. The microscope shows that complete destruction of its secreting cells occurs, so that in the most severe cases not a single cell has been left entire; dark masses of biliary pigment, hæmatine, and fat alone remain; and the kidneys are found gorged with new products. The redder portions of the liver are more destroyed than the yellow, which show remains of cells containing bile. The symptoms set in, like a bilious attack, with feebleness, rapidly followed by jaundice, febrile symptoms, and vomiting,

delirium supervening by the third or fourth day. Convulsive fits soon pass into unconsciousness, associated with stertorous breathing, foaming at the mouth, clenched teeth, closed eyes, with pupils normal and susceptible. The pulse is at first slow, but at the outbreak of the nervous symptoms it gradually rises to 110 or 120, and presents remarkable variations as regards frequency and volume. Towards the close of the disease it increases in frequency, and becomes smaller and smaller till it can no longer be felt. The symptoms generally resemble those of uræmic intoxication. The extent of hepatic dulness diminishes more and more as the disease advances, and not unfrequently the dull space disappears completely, without any tympanitic distension of the bowels to cause the dull sound over the liver to be obscured. The spleen, at the same time, is increased in volume. The bowels are almost always confined; and the stools are firm, dry, clay-like, deficient in bile, and at a later period not unfrequently dark-coloured and tarry, from the presence of blood. The urine is more or less saturated with a brown colouring matter, presents the re-action of bile-pigment, and deposits a light or greenish-yellow precipitate, in which the microscope detects the epithelium of the passages and of the kidney coloured yellow, and *needle-shaped crystals* (*tyrosine*) covered with colouring matter, either isolated or adhering in crystalline masses (FRERICHS). Sometimes the urine ceases to pass, and may be retained in the bladder as a clear bilious fluid, having a specific gravity of 1030 (WILKS); or ranging from 1012 to 1024, always of acid re-action, and occasionally slightly albuminous for short periods (FRERICHS). The evaporation of the urine gives crystals of *leucine*, which are in round masses, and of *tyrosine*, which are needle-shaped. Sometimes pure tyrosine falls as a sediment. These crystals are also found in the substance of the liver, but only in the hepatic vein, not in the portal vein nor hepatic artery (FRERICHS). The question as to retention of urea, or its lessening as due to a want of complete metamorphosis of albuminous products, is not yet determined (PARKES, l. c., p. 286).

The disease runs a more or less violent and rapid course. In severe cases it has terminated at the end of twelve or twenty-four hours (FRERICHS). In other cases in from two to eight days, urgent symptoms ending the disease by extreme prostration or excessive vomiting (WILKS). It is scarcely ever prolonged to a

week, except when it occurs after jaundice, when the whole duration of the disease may extend over four to five weeks. After the commencement of the characteristic symptoms, the disease almost invariably terminates in five days; and as a rule the termination has been fatal to life. The prognosis is, therefore, in the highest degree unfavourable. Hæmorrhages are apt to occur (in one-half the cases) simultaneously from various parts of the body, usually from the stomach, bowels, or uterus. *Post-mortem* examination shows also extravasation upon mucous and serous membranes.

Pressure over the region of the liver gives rise to marked indications of pain, which are expressed even during coma.

Morbid Anatomy.—The size of the organ is found after death to be diminished in all directions, but especially in its thickness. The gland is flattened out. The parenchyma is flabby and shrivelled. Where the disease has advanced the farthest, the cut surface has an ochre-yellow or rhubarb-like colour; and the blood-vessels are empty, and the traces of the outlines of lobules are obliterated.

At places where the process is less advanced extravasations exist, or their remains, in the forms of crystals of hæmatoidine. The lobules are encircled by congested vessels; and a dirty yellow substance separates them from each other. Afterwards the capillary congestion recedes, the size of the lobules diminishes, their colour becomes yellower, whilst the intervening grey substance gradually increases.

Bright has recorded the weight of such livers as reduced to *two pounds*, or to *twenty-three ounces*, and even to *nineteen ounces*. Frerichs records the weight of one as low as *one pound thirteen ounces;* its relative weight to the entire body being as 1 to 68 or 54—*i. e.*, a reduction of more than one-half.

Causes.—The mode of origin of acute atrophy of the liver is still unknown; but the circumstances under which the affection has occurred may be stated as follow:—Mental distress, dissolute habits, venereal excesses, syphilis, drunkenness, nervous depression, pregnancy, and the influence of miasmata. Looking to the occurrence of albumen in the urine of pregnant women, it may be that atrophy of the liver and the *nephritis* of pregnancy are both due to some condition of blood which in certain states of the constitution may be induced by pregnancy (Wilks). Atrophy of

the liver has been most frequently observed in females; and more than half were attacked during pregnancy; and the period of life between twenty to thirty years of age seems most predisposed to the disease.

Acute atrophy of the liver is apt to be mistaken for typhus fever, complicated with jaundice or with pyæmia. The range of temperature may help to indicate the disease, inasmuch as the temperature is said not to be increased in cases of acute yellow atrophy (see FRERICHS, vol. i., p. 217).

Treatment.—English physicians recommend emetics and purgatives (CORRIGAN, GRIFFIN, HANLON). Frerichs recommends that the stronger purgatives should be used simply to remove congestion, such as *senna, aloes, colocynth;* and the doses should be such as to secure profuse evacuations. When atrophy has distinctly commenced, no benefit results from medicinal treatment. Severe pains indicate the use of leeches, cupping, and cold cloths, or fomentations, as recommended under typhoid fever in vol. i. The occurrence of hæmorrhage indicates the use of mineral acids.

SECTION V.—DISEASES IN WHICH LESIONS TEND TO BE LOCALIZED IN THE KIDNEYS.

A careful study of the structure and functions of the kidney is of the greatest importance to the student of medicine. The determination of the quality and quantity of the *excreta* eliminated in the form of urine is indeed one of the best methods for enabling him to determine not only morbid conditions of the kidney, but to appreciate many changes which go on in the body during disease—changes which have frequently been referred to throughout these volumes. In every case of disease (in wasting, febrile, and constitutional diseases especially) much may be learned regarding their natural history by a careful examination of the urinary excretions, and of the microscopic characters of the sediment, especially when studied in connection with the ranges of temperature of the body, the number of respirations, and the state of the pulse, during daily periods of twenty-four hours. A formula has been already suggested at page 101 as a method to be followed in daily records; and it is now clearly understood that the *absolute amount excreted in a given time, and referring that amount to body-weight,* are the only conditions which will

yield anything approaching to accurate scientific results (ROUTH, PARKES). "In a medical point of view it is a mere waste of time to estimate the constituents in a certain quantity of urine, passed at any particular hour of the day or night, without ascertaining the relation which that quantity, with its constituents, bears to the whole quantity passed during the *twenty-four* hours; and this is the more necessary as the amount of fluid secreted varies considerably in healthy persons (SUTTON). The volumetric methods of determining the urinary constituents have greatly facilitated such investigations; and these methods are clearly expounded in the works of Hoppe, Neubaur, and Vogel, Thudichum, Beale, Hassall, and Sutton; and, above all, the profession is indebted to Dr. Parkes for his philosophical exposition of the value of our present knowledge regarding the state of the urine in health and disease, as set forth in his admirable work on that subject, already frequently referred to. From these works the following sections are compiled.

SECTION VI.—ON DETERMINING THE COMPOSITION OF THE URINE IN DISEASE.

It is examined for two purposes,—(1.) To discover the condition of the urinary organs; (2.) To determine the course of the abnormal metamorphoses of tissue in the body which lead to alterations in the composition of the several excreta.

The normal urinary constituents have probably a fixed physiological range concurrent with age and weight of the body, so that a person ought not to continue passing regularly an amount of any constituent very greatly above or below the limits of the range proper to him, otherwise some morbid condition must be sought for to explain the nature of the occurrence. The averages of the excreta for at least ten days should be taken in health, in order to arrive at an accurate standard. But as this is not possible in cases of disease, certain empirical formula are laid down by Dr. Parkes for calculating the urinary excretion in a sick person whose normal excretion is unknown. The following are the details of the facts to be determined and recorded:—

I. Record the age and weight of the patient in pounds avoirdupois.

II. Collect all the urine passed in twenty-four hours; measure it in cubic centimetres, and record its absolute amount.

III. Observe and record as to the following general properties, viz.:—(1.) Specific gravity; (2.) Uræmatine, as determined by Vogel's colour table;* (3.) Clearness or turbidity on emission or after rest; (4.) Determine the absolute weight, by multiplying the quantity passed, expressed in c. c., by the figures expressing the specific gravity,—the result is the weight in grammes.

IV. Set aside the following quantities for the volumetric determination of—

1. Urea,	40 c. c.
2. Uric Acid,	300 to 500 c. c.
3. Phosphoric Acid,	40 c. c.†
4. Chloride of Sodium,	40 c. c.
5. Sulphuric Acid,	100 c. c.
6. Degree of Free Acidity—	
7. Sugar,	20 c. c.
8. Albumen—	
Solids,	20 grammes.

V. Collect and examine the sediment.

VI. Determine the amount of excretion normal to the individual by the following empirical formula (PARKES):—Multiply the following figures by the weight of the person in pounds avoirdupois; the result is the excretion in grains, in twenty-four hours, of the several ingredients of the urine:—

* Vogel's colour table is made by taking certain colours as starting-points, and representing the following groups for comparison:—

I. *First group; yellowish urines*, represented by gamboge mixed with a greater or less amount of water, giving three grades, the first of which is almost colourless, as follow:—(1.) *Pale yellow* (gamboge with much water); (2.) *Light yellow* (gamboge with less water); (3.) *Yellow* (gamboge with very little water).

II. *Second group; reddish urines*, represented by the above yellow grades mixed with a greater or less amount of red—*i. e.*, gamboge with crimson lake in three grades, as follow:—(4.) *Reddish-yellow* (gamboge with a little crimson lake); (5.) *Yellowish-red* (gamboge with more crimson lake); (6.) *Red* (crimson lake with little gamboge).

III. *Third group; brown or dark urines*, represented by gamboge, crimson lake, and more or less Prussian blue, in three grades, as follow:—(7.) *Brownish-red* (red with an admixture of a little brown); (8.) *Reddish-brown* (more of the brown than in the last); (9.) *Brownish-black* (almost black, with a touch of the reddish-brown) (see Thudichum *On the Pathology of the Urine*, p. 134).

† The precipitate from the urea estimation is sufficient.

	In Men between 20 and 40.	In Women between 20 and 40.	In Children between		In Young Men and Women between 16 and 20.
			3 and 8.	8 and 16.	
Urea, ...	3·53	2·96	6·83	5·20	4·39
Chlorine, ...	0·875	0·817	1·44	1·097	0·926
Sulphuric Acid, . .	0·214	0·25	0·414	0·315	0·266
Phosphoric Acid, .	0·336	0·336	0·65	0·495	0·418

The following corrections are required:—(1.) If the person be between forty and fifty, calculate according to columns 1 or 2, and then deduct 10 *per cent.;* for ages between fifty and sixty deduct 20 *per cent.;* for ages between sixty and seventy deduct 30 *per cent.;* for ages upwards of seventy deduct 50 *per cent.* (2.) If the person has been starving for two or more days (as in some fevers), deduct one-third from the calculation made according to the table; if the diet be meagre, deduct one-eighth or one-sixth; if pretty plentiful, yet still below that of health, deduct one-tenth. (4.) If there be total inactivity, deduct one-tenth; if there be merely quietude, deduct one-twentieth.

Section VII.—Volumetric Estimation of the more Important Constituents of the urine; and their Pathological Relations.

The Volumetric Method of Analysis is one which enables ordinarily skilful operators, or medical men who cannot devote much time to practical chemistry, to determine with sufficient accuracy the amount of the most important constituents of the urine, such as *urea, uric acid, chloride of sodium, phosphates, sulphates,* and the *free acidity* of this excretion. Certain conditions are necessary for success, namely,—(1.) Solutions of the re-agents or tests, the composition and strength, or chemical power of which are accurately known; (2.) *Burettes,* graduated tubes or vessels from which portions of the test-solutions may be accurately delivered; (3.) The power of determining by the eye when the decomposition produced by the test-solution with the urine has ceased, so that the quantity of test-solution used (*i. e.,* with which

certain components of the urine have combined) may be accurately determined.*

It is necessary to exercise the greatest care in the graduation of the measuring instruments, and in the strength and purity of the standard solutions; because a very slight error in the process is greatly magnified—in proportion, in fact, as it is multiplied to represent the amount in large quantities when the actual observation has been made upon a small amount.

1. **Estimation of Chlorides.**—They are calculated as *chloride of sodium;* the test-solutions required being—(1.) The *standard solution of the nitrate of mercury;* and (2.) The "baryta-solution," as it is commonly called. The method was devised by Liebig, and its principle is as follows:—"If the solution of nitrate of mercury, free from any excess of acid, is added to a solution of urea, a *white gelatinous precipitate* is produced, containing urea and oxide of mercury in the proportion of 1 eq. of the former to 4 eqs. of the latter. But when *chloride of sodium* is present in the solution, *the precipitate does not occur until all the chloride of sodium is converted into chloride of mercury* (*sublimate*) *and nitrate of soda,* the solution *remaining clear.* If the exact point be overstepped, the excess of mercury immediately produces the precipitate above described, so that the *urea* present acts as an indicator of the end of the process. It is therefore easy to ascertain the proportion of chlorides in any given sample of urine by this method, if the strength of the mercurial solution is known; since 1 eq. of oxide of mercury converts 1 eq. of chloride of sodium into 1 eq. each of corrosive sublimate and nitrate of soda" (SUTTON).

The steps of the process are as follow:—

(1.) Take 40 c. c. of urine; (2.) Mix with 20 c. c. of the baryta-solution; (3.) Pour the thick mixture upon a small dry filter; and when sufficient clear liquid has passed through, (4.) Take 15 c. c. of it (= 10 c. c. of urine), and just neutralize it, or render it acid by a drop or two of nitric acid; (5.) Bring this urine fluid under the burette which contains the test-solution of the nitrate of mercury, which is to be allowed to drop gradually, drop by drop, into the beaker containing the urine, which is

* The apparatus and the test-solutions required, graduated to the proper strengths, are supplied by Mr. Griffin, 119 Bunhill Row; and by Messrs. Bullock and Reynolds, Hanover Street, London; also Harper and Sutton, Operative Chemists, Norwich.

Geographical Distribution of HEALTH AND DISEASE

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ARCTIC OCEAN
ATLANTIC OCEAN
PACIFIC OCEAN
INDIAN OCEAN
ANTARCTIC OCEAN
EUROPE
ASIA
NORTH AMERICA
SOUTH AMERICA
AFRICA
AUSTRALIA
GREENLAND
BAFFIN BAY
Arctic Circle
Equator
Tropic of Capricorn
Long. W. of 60 Greenwich
Long. E. of 80 Greenwich
Explanation of Colours
Torrid Zone of Disease
Sub-Torrid & Temperate
Sub-Temperate & Arctic
Small Pox
Leprosy
Goitre
Catarrh
Cholera
Yellow Fever
Plague
Ague
Elephantiasis
Cretinism & Goitre
Rheumatism Prevalent
Very Healthy
Very Unhealthy
Very Healthy
Very Unhealthy
Very Healthy
Very Unhealthy

to be constantly stirred with a glass rod; (6.) As soon as the precipitate does not disappear by stirring, the operation is finished, but a permanent precipitate is produced (= urea and oxide of mercury); (7.) The volume of the test-solution used is to be read off the burette, and the amount of *chloride of sodium* calculated therefrom; (8.) The chlorine may be estimated by the following formula:—As 58·8 eqs. of chloride of sodium contain 35·5 eqs. of chlorine, the chlorine in the urine is obtained by the equation—

58·8 : 35·5 : : amount of chloride of sodium in the urine : x (the chlorine it contains).

The average amount of chlorine excreted in twenty-four hours is about 8·21 grammes, or 126·76 grains; 13·6 grammes, or 210 grains, of chloride of sodium, were the chloride always united with that substance—an amount which Dr. Parkes thinks is too great. Vogel and Parkes consider the mean to be 7 grammes, or 108 grains, = 11·5 grammes, or 177 grains, of chloride of sodium in twenty-four hours; the range above and below the mean being from 30 to 60 *per cent.*

In relation to body-weight, the averages of chlorine are as represented in the second line of the table at p. 911, paragraph VI.

Pathological Relations.—The chlorine contained in the urine is wholly derived from the food; and a part, chiefly united with *sodium*, passes out of the system without having entered into the composition of the tissues; another portion, uniting with the tissues, is only set free on the disintegration of these tissues; and therefore, whatever exceeds the amount taken in by the food is derived from and represents change of tissue. In *ague* its elimination is increased during the cold and hot stages. In *acute pneumonia* their elimination is greatly lessened, often entirely absent, but re-appearing in the urine eight or ten days after resolution has set in and excess of urea has passed away. It evidently accumulates in the inflamed portion of the lungs. Their amount is also reduced in *typhus and typhoid fever, febricula, scarlatina, erysipelas, puerperal fever, pleurisy, acute capillary bronchitis, acute pulmonary phthisis, acute rheumatism, cholera, acute and chronic Bright's disease.*

Treatment.—Elimination is to be effected by the skin, bowels, or urine, mainly by the action of *liquor potassæ.*

2. **The Estimation of Urea** in urine is based on the combination which forms between urea and oxide of mercury in neutral or

alkaline solutions. The method was devised by Liebig. The precipitate which is formed is insoluble in water, or in weak alkaline solutions. The standard test-solutions are the same as in the preceding estimate; and the indicator which shows when all the urea has entered into combination with the mercury, and when the latter predominates, is a solution of carbonate of soda.

(1.) Take one volume of the baryta-solution (20 c. c.), and mix with two volumes of urine (40 c. c.);* (2.) After filtration take 15 c. c. of the fluid (= 10 c. c. of urine) for each analysis, in small beakers; (3.) Bring the beaker under the burette containing the mercurial solution, which is to be added in small quantities so long as a distinct precipitate is seen to form, the mixture being stirred constantly; (4.) A plate of glass or porcelain is now to be sprinkled with a few drops of solution of carbonate of soda, and a drop of the mixture brought from time to time in contact with the drops of soda-solution by means of a glass rod. So long as the mixture of the two drops thus brought in contact remains white, free urea is still present in the mixture, and more of the test-solution must be added to the urine, till the contact of the drops with the soda-solution produces a yellow colour, which is distinctly apparent; (5.) Record the quantity of mercurial test-solution used, and so calculate for the amount of urea contained in the 10 c. c. of urine, and hence in the total discharge for twenty-four hours; (6.) Repeat the analysis at least twice.

Another method has been recommended by Dr. Edmund W. Davy (*Phil. Mag.*, vol. vii., Fourth Series, p. 385). The process is based on the fact that *urea* is readily decomposed by *hypochlorite of soda*, when the *nitrogen* being evolved as a gas, the amount of urea is estimated from the amount of nitrogen gas produced by the decomposition. A strong glass tube is required, about fourteen inches long, closed at one end, and its open extremity ground smooth. The bore of the tube must not be larger than the thumb can conveniently cover—*i. e.*, half an inch in diameter. It ought to be graduated into cubic inches, commencing from the closed end, and each cubic inch again subdivided into 10ths and 100ths.†

The following are the details of the process:—

(1.) Fill the tube more than one-third full of fluid mercury; (2.) Pour in carefully half a fluid drachm to one drachm of urine; (3.) Holding

* The precipitate may be reserved for determining the phosphoric acid.

† Cassela, 23 Hatton Garden; Negrette & Co., Holborn Hill; also Messrs. Griffin, 119 Bunhill Row, London, keep such tubes.

the tube in one hand near its open extremity, and having the thumb in readiness to cover the aperture, quickly fill it completely full, with a solution of hypochlorite of soda (taking care not to overflow the tube), and then instantly cover the opening tight with the thumb; (4.) Rapidly invert the tube once or twice to mix its contents; and (5.) Finally open the tube under mercury contained in a strong cup or small mortar; (6.) The tube is left in the upright position till the evolution of gas ceases, which it generally does in from three to four hours; (7.) Record the amount of gas found, and estimate the urea by the following data:—1·549 cubic inch of gas represents one grain of urea.

The hypochlorite of soda used should always be five or six times the volume of the urine operated upon; and the *liquor sodæ chlorinatæ* should be perfectly pure, prepared according to the process of the Dublin Pharmacopœia, as the *liquor* of commerce always gives erroneous and exaggerated results.

A third method for determining the urea consists in the use of the following table, which is founded on many observations of urine, both of health and disease, of specific gravities from 1,003 to 1,028. The Rev. Samuel Haughton has devised this table, and has given in its results, which he believes will prove very useful, approximations to the daily excretion of urea, in all cases in which sugar is absent, and albumen either absent or only present in small quantities (*Medical Times, Oct.* 22, 1864).

The following table is one of double entry, to be used by finding the daily excretion of urine in fluid ounces, and its specific gravity, by means of a carefully graduated urinometer. When these data are found, at the intersection of the corresponding columns, the excretion of urea is given in grains. "I submit it with confidence," says Mr. Haughton, "to the test of practical experience, as I have so often used it myself in fever, in pneumonia, in dyspepsia, and in kidney diseases, that I believe it will be found a valuable aid to the physician, both in the prognosis and in the treatment of these and other diseases.*

* My friend Dr. De Chaumont has compared the trustworthiness of these processes in twenty-six cases (mostly healthy, and none either albuminous or diabetic), with the following results:—"By Liebig's process the actual average gave 25·78 parts in 1,000. The average of nineteen of these taken from Haughton's Table (the remaining seven being of specific gravity above 1,028, the limit of Haughton's Table) gave 28·17 parts in 1,000. The average of the whole twenty-six, deduced from Haughton's second formula (viz., *excess* of *sp. gr. over* 1,000 × 10 = grs. in a pint) gave 32·81 parts in 1,000.

[*Continued on p.* 918.

Fluid Ounces.	SPECIFIC GRAVITY.																								
	1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028
20	35	36	43	57	71	85	100	103	106	119	130	136	142	151	160	196	233	241	249	257	265	274	276	278	279	280
21	37	38	45	59	74	89	105	108	111	124	136	142	149	158	168	205	245	253	261	269	278	288	290	292	292	294
22	38	40	47	62	78	95	110	113	116	130	143	149	156	166	176	215	257	265	274	282	292	301	303	305	306	308
23	40	41	49	65	81	97	115	118	121	136	149	156	163	173	184	225	268	277	286	295	305	315	317	319	320	322
24	42	43	51	68	85	101	120	123	127	142	156	163	170	181	192	235	280	289	299	308	319	329	331	333	334	336
25	43	45	53	71	88	106	125	129	132	147	162	170	177	188	200	245	291	301	311	321	332	342	345	347	348	350
26	45	47	55	73	92	110	130	134	137	153	169	176	184	196	208	254	303	313	324	334	346	356	359	360	362	364
27	47	49	57	76	95	114	135	139	143	159	175	183	191	213	216	264	314	325	336	347	359	369	372	374	376	378
28	48	50	59	79	99	118	140	144	148	165	182	190	198	221	224	274	326	337	349	360	372	383	386	388	390	392
29	50	52	61	82	103	122	145	149	153	171	188	197	205	228	232	284	337	349	361	373	386	397	400	402	404	406
30	52	54	64	85	106	127	150	155	159	177	195	204	213	226	240	294	349	361	374	386	399	411	414	416	418	420
31	54	55	66	87	109	131	155	160	164	182	201	210	220	233	248	303	361	373	386	398	412	425	428	429	432	434
32	55	57	68	90	113	135	160	165	169	188	208	217	227	241	256	313	373	385	398	411	425	438	442	443	446	448
33	57	59	70	93	116	140	165	170	175	194	214	224	234	249	264	323	384	397	411	424	438	452	455	457	460	462
34	58	61	72	96	120	144	170	175	180	200	221	231	241	256	272	333	396	409	423	437	451	466	469	471	474	476
35	60	63	74	99	124	148	175	180	185	206	227	238	248	264	280	343	407	421	436	450	464	479	483	485	488	490
36	61	64	76	102	127	153	180	185	191	212	234	244	255	271	288	352	419	433	448	462	477	493	497	499	502	504
37	63	66	78	105	130	157	185	190	196	218	240	251	262	279	296	362	430	445	461	475	490	507	510	513	516	518
38	65	68	80	108	134	161	190	195	201	224	247	258	269	286	304	372	442	457	473	488	503	520	524	527	530	532
39	67	70	82	111	138	166	195	200	206	230	253	265	276	294	312	382	453	469	486	501	516	534	538	541	544	546
40	69	72	85	114	142	170	200	206	212	236	260	272	284	302	320	392	465	482	498	514	530	548	552	555	558	560
41	71	73	87	116	145	174	205	211	217	241	266	278	291	309	328	401	477	494	510	527	543	562	566	568	571	574
42	74	75	89	119	148	178	210	216	222	247	273	285	298	317	336	411	489	506	523	540	557	575	580	582	585	588
43	75	77	91	122	152	182	215	221	228	253	279	292	305	324	344	421	500	518	535	553	570	589	593	596	599	602
44	76	79	93	125	156	186	220	226	233	259	286	299	312	332	352	431	512	530	548	566	584	603	607	610	613	616
45	78	81	95	128	160	191	225	231	238	265	292	306	319	339	360	441	523	542	561	579	597	616	621	624	627	630
46	80	82	97	130	163	195	230	236	243	271	299	312	326	347	368	450	535	554	573	592	611	630	635	638	641	644
47	82	84	99	133	166	199	235	241	249	277	305	319	333	355	376	460	546	566	586	605	624	644	648	652	655	658
48	84	86	101	136	170	203	240	246	254	283	312	326	340	362	384	470	558	578	598	618	637	657	662	666	669	672

49	85	88	103	139	174	207	245	251	259	289	318	333	347	370	392	480	569	590	611	631	651	671	676	680	683	686
50	87	90	106	142	178	212	250	257	265	295	325	340	355	377	400	490	581	602	623	644	665	685	690	694	697	700
51	88	92	108	144	181	216	255	262	270	301	331	346	362	385	408	499	593	614	635	656	678	699	704	708	710	714
52	90	94	110	147	185	220	260	267	276	307	338	353	369	393	416	509	605	626	648	669	692	712	718	721	724	728
53	92	96	112	150	188	225	265	272	281	313	344	360	376	400	424	519	616	638	660	682	705	726	731	735	738	742
54	94	98	114	153	192	229	270	277	286	319	351	367	383	408	432	529	628	650	673	695	718	740	745	749	752	756
55	95	99	117	156	195	233	275	283	292	325	358	374	390	415	440	539	639	662	685	708	732	753	759	763	766	770
56	96	100	119	159	199	238	280	288	297	331	364	380	397	423	448	548	651	674	698	720	745	767	772	776	780	784
57	98	102	121	162	202	242	285	293	303	337	371	387	404	430	456	558	662	686	710	733	758	781	786	790	794	798
58	100	104	123	165	206	246	290	298	308	343	377	394	411	438	464	568	674	698	723	746	772	794	800	804	808	812
59	102	106	125	168	209	251	295	303	314	349	384	401	418	445	472	578	685	710	735	759	785	808	814	818	822	836
60	104	108	128	171	213	255	300	309	319	355	391	408	426	453	480	588	697	722	748	772	798	822	828	832	836	840
61	106	109	130	173	216	259	305	314	324	360	397	414	433	460	488	597	708	734	760	784	811	836	842	845	850	854
62	108	110	132	176	220	263	310	319	329	366	404	421	440	468	496	607	719	746	772	797	824	849	856	859	864	868
63	109	112	134	179	223	267	315	324	335	372	410	428	447	475	504	617	730	758	785	810	838	863	869	873	878	882
64	110	114	136	182	227	271	320	329	340	378	417	435	454	483	512	627	742	770	797	823	851	877	883	887	892	896
65	112	116	138	185	230	276	325	335	345	384	423	442	461	490	520	637	754	782	810	836	864	890	897	901	906	910
66	114	118	140	187	234	280	330	340	351	390	431	448	468	498	528	646	766	794	822	849	877	904	911	915	920	924
67	115	120	142	190	237	284	335	345	356	396	437	455	475	505	536	656	778	806	835	862	891	918	925	929	934	938
68	116	122	144	193	240	288	340	350	361	402	443	462	482	513	544	666	790	818	847	875	904	931	939	943	948	952
69	118	124	146	196	244	292	345	355	367	408	449	469	489	520	552	676	802	830	860	888	917	945	953	957	962	966
70	120	126	149	199	248	297	350	361	372	414	456	476	497	528	560	686	814	843	872	901	930	959	966	971	976	980
71	121	127	151	201	251	301	355	366	377	419	462	482	504	536	568	695	826	855	884	913	943	973	980	984	990	994
72	122	128	153	204	255	305	360	371	382	425	469	489	511	544	576	705	838	867	896	926	956	986	994	998	1004	1008
73	124	130	155	207	258	310	365	376	388	431	475	496	518	551	584	715	849	879	909	939	969	1000	1007	1012	1018	1022
74	126	132	157	210	262	314	370	381	393	437	482	503	525	558	592	725	861	891	921	951	982	1014	1021	1026	1032	1036
75	128	134	159	213	266	318	375	386	398	443	488	510	532	566	600	735	872	903	934	964	995	1027	1035	1040	1046	1050
76	130	136	161	216	269	323	380	391	404	449	495	516	539	573	608	745	884	915	946	977	1008	1041	1049	1054	1060	1064
77	132	138	163	219	273	327	385	396	409	455	501	523	546	581	616	755	895	927	959	989	1021	1055	1062	1068	1074	1078
78	134	140	165	222	276	331	390	401	414	461	508	530	553	588	624	765	907	939	971	1002	1034	1068	1076	1082	1088	1092
79	136	142	167	225	280	336	395	406	420	467	514	537	560	596	632	775	918	941	984	1015	1047	1082	1090	1096	1102	1106
80	139	144	170	228	284	340	400	412	425	473	521	544	568	604	640	785	930	964	996	1028	1060	1096	1104	1110	1116	1120

	1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028

The mean amount of urea excreted by adult males between twenty and forty years of age during the twenty-four hours is 33·18 grammes, = 512·4 grains. If the mode of life be equable, the amount remains pretty constant; but the maximum and minimum amounts passed on any one day by an individual are usually about *one-fifth* above or below the mean amount. In relation to body-weight, the average excretion of *urea* is as stated in the first line of the table at p. 911, paragraph VI.

Pathological Relations.—All the nitrogenous tissues contribute, by their disintegration, to the formation of urea; and the urea excreted may be taken as a measure of the extent of tissue-change or waste. In all *pyrexial states* its formation is increased; but its elimination may be increased or diminished, (see chapter on "Fever," vol. i.) When freely eliminated by the urine, its amount stands in close relationship with the exaltation of temperature and intensity of the fever. Retention of urea is a very unfavourable circumstance. No retention occurs in *acute yellow atrophy of the liver*, in *epilepsy*, in *jaundice*.

There are some cases, which stand by themselves, in which the excretion and elimination of urea is augmented, which were originally noticed by Prout, and which Willis subsequently described as cases of "*azoturia*." They have recently been noticed in an excellent paper by Dr. Sieveking in the pages of the *British Medical Journal* for May, 1865. They are characterized by a great excess of urea in the urine, without increase in the quantity of the urine, and apparently without febrile re-action, but with languor, weakness, and nervousness, associated sometimes with

Note—Continued from p. 915.]

The average obtained by the formula A:—*Excess of sp. gr. over* 1,000 × 10 = parts in 1,000, gave 28·71 parts in 1,000.

The average obtained by the formula B:—*Excess of sp. gr. over* 1,000, *after elimination of the sp. gr. due to chlorides*, ÷ ·5 (= × 2) = parts in 1,000, gave 26·200 per 1,000.

The average obtained by the formula C:—*Excess of sp. gr. over* 1,000, *after elimination of sp. gr. due to chlorides*, ÷ ·52 = parts in 1,000, gave 25·65 parts in 1,000.

Tabulating these, we have:—

True Average, ...	= 25·78	*error.*
Haughton's Table, ...	= 28·17	+ 9·2 *per cent.*
Haughton's Second Formula, . . .	= 32·81	+ 23·4 ,,
Dr. De Chaumont's Formula A, . . .	= 28·71	+ 11·3 ,,
Dr. De Chaumont's Formula B, . . .	= 26·203	+ 1·6 ,,
Dr. De Chaumont's Formula C, . . .	= 25·650	— ·5 ,,

dyspepsia, intemperance, mental anxiety, or sexual excess. They are apt to pass into *diabetes mellitus* (PROUT).

3. **Estimation of Sulphuric Acid** requires the *chloride of barium* test-solution and Beale's filter. The details of the process are as follow:—

(1.) Measure off 100 c. c. of urine into a Florence flask; (2.) Add a little hydrochloric acid (twenty or thirty drops); (3.) Apply heat through a sand bath till the acidulated urine boils; (4.) Allow the chloride of barium test to flow very gradually into the urine; (5.) Remove the heat, and allow the precipitate to subside after each addition; (6.) Continue adding the test till the precipitation is complete; (7.) Use Beale's filter for ascertaining the end of the precipitation. When no precipitate is formed either by chloride of barium, or by sulphate of potassa or soda, the analysis is complete.

The mean of sulphuric acid excreted in twenty-four hours is 2·012 grammes, = 31·11 grains, with a range of 45 *per cent.* above or below. In relation to body-weight, the average excretion of sulphuric acid is as stated in the third line of the table at page 911, paragraph VI.

Pathological Relations.—Sulphuric acid originates in the urine from various articles of food and drink, in which it exists ready formed: from the sulphur of the food, by oxidation in its passage through the system; from oxidation of the sulphur of the tissues; or from the oxidation of sulphur contained in substances such as *taurine* and *cystine*. Its elimination is increased in *febricula, typhoid fever, typhus, variola, pyæmia, milk fever, delirium tremens, acute pneumonia, rheumatic fever, chorea.*

4. **Estimation of Phosphoric Acid** is based on the fact, that when nitrate or acetate of uranium is added to a solution of tribasic phosphoric acid, containing acetate of ammonia and free acetic acid, the whole of the phosphoric acid is thrown down as double phosphate of uranium and ammonia, having a light-brown colour and a composition represented by the formula $2\,(Ur_2\,O_3)$, $N\,H_4\,O$, $PO_5 + Aq$. This volumetric method for the estimation of phosphoric acid was devised by Mr. Francis Sutton, of Norwich, independently of Neubauer and Pincus, who, independently of each other, also arrived at the same process; but Mr. Sutton states that Neubauer was the earliest discoverer of the method. The standard test-solution required is the nitrate of uranium, of which 1 c. c. represents 0·1 grain of phosphoric acid. A solution of ferro-

cyanide of potassium (yellow prussiate of potash), in the proportion of 1 to 20, is to be used as an indicator to stop the process, so soon as the re-action is complete.

The details of the process are as follow:—

(1.) Take the precipitate produced by the baryta-solution in 40 c. c. of urine, as mentioned at page 911, paragraph VI., having been set aside after the fluid is filtered from it. (2.) Wash the precipitate once with cold water. (3.) Treat it, while still on the filter, with warm acetic acid, to dissolve all the phosphate of baryta, which passes through the filter, leaving the sulphate behind. (4.) Wash the filter with a small quantity of boiling water, so as to remove the last traces of phosphate. (5.) Add sufficient ammonia to the solution to neutralize the acetic acid, unless the quantity of the latter be large, when somewhat less than enough to neutralize may be added. Under any circumstances the liquid must be fully acidified with acetic acid before being tested as to its strength (*titrated*), and must contain a tolerable quantity of acetate of ammonia. (6.) Take a measured quantity of the urinary phosphate-solution (say 20 c. c.) in a beaker, and gently warm it, and bring it under the burette containing the uranium-solution. (7.) Portions of the uranium-solution are to be delivered in and constantly stirred, until, when a drop is taken out with a thin glass rod, and placed in the middle of a large drop of the solution of yellow prussiate of potash on a white plate, a faint but distinct chocolate-brown colour is produced at the point of contact. A slight excess of uranium-solution is required to produce the brown colour, which indicates that all the phosphoric acid contained in the amount of urine in the beaker has been thrown down. (8.) Record the amount of uranium-solution used, and estimate accordingly—1 c. c. of the solution precipitating 0·1 grain of phosphoric acid.*

The mean amount of phosphoric acid excreted in twenty-four hours is 3·164 grammes, = 48·80 grains, with a range in the same person of from 35 to 50 *per cent.* The amount in relation to body-weight is given in the fourth line of the table at page 911, paragraph VI.

Pathological Relations.—Its elimination in excess represents tissue-change or waste, beyond what the food or drink accounts for. In rickets and softening of the bones the phosphates are increased.

* Collect all the precipitates in a large bottle, and when sufficient has been obtained, recover the uranium by igniting the dry precipitate in a porcelain crucible, with the carbonaceous residue produced by burning tartrate of soda and potash in a covered crucible. The uranium is thus reduced to protoxide, while the phosphoric acid unites with potash and soda, and can be entirely extracted with boiling water. The protoxide of uranium left may be dissolved in nitric acid, and evaporated to dryness in a water-bath (Sutton, *Volumetric Analysis*, p. 208).

In other diseases our information respecting phosphoric acid is very uncertain. In alkaline urine the *earthy* phosphates are deposited as a white sediment. Mineral acids, anodynes, alkaline bicarbonates, opium, and preparations of iron, zinc, and strychnia, are often of service, variously combined, according to the nature of the case (see HASSALL, p. 237).

5. **The Estimation of Uric Acid.**—An improved method of estimating the uric acid by iodine has been recently devised by my friend Dr. De Chaumont, and published by him in the *Medical Report of the Army Medical Department* for 1862 (published in 1864). The determination of uric acid by iodine is founded on the fact that solution of iodine is decolorized by uric acid, in a definite proportion—which appears to be four equivalents to one of uric acid. Dr. De Chaumont has kindly given me the following short notice of the method:—

The Solutions required are as follow:—

(1.) A standard solution of iodine. The alcoholic tincture being liable to change, it is best to dissolve the iodine in water with the aid of iodide of potassium; thus, 6·35 grammes (= $\frac{1}{20}$th of an equivalent) of iodine, with 12 grammes of iodide of potassium, are dissolved in a litre of distilled water. Of this solution, 1 c. c. contains ·00635 of iodine, and will decompose ·0021 of uric acid. If the materials be accurately weighed and measured, it will be unnecessary to graduate the solution; but, should this be desired, then the following solutions must be prepared:—(2.) Solution of uric acid, ·168 grammes are to be dissolved in a litre of water. This is best made by dissolving the uric acid in a small quantity of *liquor potassæ*, and neutralizing the excess with dilute acetic acid. Of this solution, 12·5 c. c. will completely decolorize 1 c. c. of the solution of iodine. (3.) A solution of starch, carefully filtered and free from suspended grains.

The Process.—(1.) Filter the urine from mucus. (2.) Acidify the urine, should it be alkaline, with acetic acid. (3.) Put 10 c. c. into a beaker-glass, and dilute it to 50 c. c. with distilled water. (4.) Add 5-10 c. c. of starch liquor. (5.) Drop in the solution of iodine from a burette graduated to $\frac{1}{10}$th c. c. (6.) Stir each time, and wait till blue colour disappears. (7.) Do not add more than $\frac{1}{10}$th c. c. at a time. (8.) When the blue colour has remained permanent for *an hour*, read off the number of c. c. of iodine used, and multiply by 21, which will give the quantity per litre.

Example.—10 c. c. of urine took 3·5 c. c. of iodine,

$$3{\cdot}5 \times {\cdot}21 = {\cdot}735 \text{ grammes per litre.}$$

For a more accurate analysis, it is necessary to wait for twenty-four hours until all the uric acid is decomposed, taking care to add fresh portions of starch from time to time as it becomes converted into dextrine. A correction is then applied as follows:—(1.) Precipitate the salts with Liebig's baryta-solution (see process for urea). (2.) Filter and acidify with acetic acid. (3.) Test with iodine as before. (4.) Deduct the number of c. c. used after the baryta from the gross amount first used, and from the remainder calculate the uric acid as before.

Example.—10 c. c. of urine took 3·5 c. c. of iodine.

10 c. c. so precipitated, filtered, and re-acidified, took ·45 c. c. of iodine.

Then $3{\cdot}5 - {\cdot}45 = 3{\cdot}05$ c. c. of iodine.

$3{\cdot}05 \times {\cdot}21 = {\cdot}6405$ grammes in a litre—the net result.

The mean amount of uric acid excreted in twenty-four hours appears to be ·555 grammes, = 8·569 grains. The observations to determine the average in any individual must extend over at least *five days*. The range between the maximum and minimum amount is as great as from 20 to 30 *per cent.* (PARKES).

Pathological Relations.—Its elimination indicates metamorphoses of tissue. It increases after full, heavy, indigestible meals; and exists not alone in the spleen in health, but in still larger quantity in most of the affections, including *ague*, in which the spleen is more especially implicated. It is increased in most of the active febrile and inflammatory affections. It is greatly increased during the paroxysm of *intermittent* fevers, also in *typhoid* and *typhus* fevers. In *small-pox* and *scarlatina* it is increased; and in *pneumonia* the increase is very large. The sediments of the *urates* and of *uric acid* are most abundant from the seventh to the thirteenth day. It is increased in most cardiac affections, in hepatic cirrhosis and leucocythæmia; and is considerably increased in rheumatic fever. During attacks of gout it is lessened or absent in the urine, and present in the blood; but as soon as fever and paroxysms abate, precipitates of *urates* and of *uric acid* become abundant. Its free elimination is a favourable symptom.

In the majority of chronic affections the uric acid is lessened in elimination. When the excess in the urine is due to indulgence in animal food, its mal-assimilation and defective cutaneous excretion, the amount of animal food must be reduced, the diet regulated carefully, and the functions of the digestive organs improved and strengthened. When *lactic, acetic,* or *butyric* acids occur as products of faulty digestion, all food known to produce such a result must be avoided. The vegetable bitters—e. g., *cinchona, gentian, calumba, serpentaria,* with or without *rhubarb* and *soda,* and followed by such remedies as keep the colour free, such as *compound galbanum pill,* with *extract of colocynth and croton oil,* will be found of great service. The action of the skin must be promoted by warm clothing. Exercise in the open air must be indulged in as much as possible; while cold baths in the morning, and friction with horse-hair gloves aid the action of the skin. The best solvent is obtained by drinking freely of soft or distilled water; but alkaline remedies, like the carbonates and bicarbonates, often give relief, as well as the salts of the vegetable acids, the *acetates, citrates,* and *bitartrates,* given in sufficient doses. The occurrence of *uric acid* in excess in the diseases mentioned must be managed according to the nature of the treatment of each disease.

6. **Estimation of Diabetic Sugar.**—The principle of the process is thus explained by Mr. Sutton. It is based on the fact that although a mixture of pure sulphate of copper, tartrate of potash, and caustic soda, mixed in proper proportions, may be boiled without undergoing change; yet, if only a trace of sugar be added, a very slight warming is enough to precipitate a portion of the copper as a protoxide (Cu_2 O). It is found that one atom of pure sugar, = 180, is capable of reducing exactly 10 atoms, = 307, of oxide of copper (Cu O) to the state of protoxide. Therefore, if the quantity of copper reduced by a given solution of sugar is known, it is easy to find the quantity of sugar present (*Volumetric Analysis,* p. 210).

A standard solution of pure sulphate of copper with tartrate of potash and caustic soda is required. It is to be prepared as follows:—

(1.) 34·64 grammes, = 346·4 grains, of pure sulphate of copper, previously powdered and pressed between blotting-paper, are weighed and dissolved in 200 c. c. of distilled water; (2.) In another vessel 173

grammes, = 1,730 grains, of pure crystallized tartrate of soda and potash (Rochelle salt) are dissolved in 480 c. c. of solution of pure caustic soda (specific gravity, 1·14); (3.) The two solutions are then to be mixed, and the deep clear blue solution diluted with distilled water till the whole measures 1,000 c. c. *One* c. c. of the solution so prepared represents 0·05 grains of grape or diabetic sugar. It must be preserved for use in a dark place, and in well stoppered bottles kept full. It should bear heating when diluted with about four or five times its quantity of distilled water, without any precipitate taking place; and should always be submitted to this test before being used. If any precipitate does occur, it probably arises from the alkali having absorbed carbonic acid; and in this case the addition of a little fresh caustic soda-solution remedies the evil (SUTTON, l. c., pp. 211 and 233).

The following are the details of the process for analysis:—

(1.) 10 c. c. of clear urine are diluted by means of a measuring flask to 200 c. c. with water, and a large burette filled with the fluid; (2.) 10 c. c. of the copper solution (= $\frac{1}{2}$ grain of sugar) are then measured into a flask, or white porcelain capsule, and 40 c. c. of distilled water added; (3.) The vessel is to be arranged over a spirit-lamp under the burette, and brought to boiling; (4.) The diluted urine is then delivered in cautiously from the burette until the last traces of blue colour are removed from the copper solution, and the precipitate is of a distinct red colour; (5.) It must be remembered that the urine has been diluted twenty times; so that the quantity used, divided by twenty, will represent the amount of the original urine used; and the estimate is to be made accordingly.*

7. **Estimation of Free Acid** is measured by a solution of carbonate of soda containing 530 grains in the 10,000 grain measure, = 53 grammes in the litre; and is represented by determining how many grains of crystallized oxalic acid a certain quantity of the soda-solution will neutralize. The details of the process are as follow:—

(1.) Take 50 or 100 c. c. of perfectly fresh urine; (2.) Add from a burette a standard solution of soda in small portions at a time (say ·5 c. c.,

* Haughton recommends the following formula:—"Multiply the excess of specific gravity over 1,000 by 20,—the result is grains of sugar in a pint of urine." The average of 234 determinations by Dr. De Chaumont gave 61·06 parts in 1,000; the average by Haughton's formula gave 77·65;—error, + 27· *per cent.* Dr. De Chaumont proposes to divide excess of specific gravity over 1,000 by ·54—result, = parts in 1,000. Haughton's factor, = ÷ ·4348, or × 2·3. Dr. De Chamount's factor, = ÷ ·54, or × 1·89.

or drop by drop); (3.) After every addition test the fluid by moistening a thin glass rod or feather with the mixture, and streak it across some well-prepared violet litmus paper; when the streaks cease to become red, the analysis is complete; (4.) Estimate how much of the standard solution has been used, and express the acidity as equal to so many grains of crystallized oxalic acid.

8. **Estimation of the Total Solid Matter.**—(1.) Measure 5 c. c. into a shallow platinum or porcelain capsule; (2.) Place it beside a vessel of strong sulphuric acid under the receiver of a powerful air-pump, and keep it *in vacuo* till all moisture is removed.

9. **Estimation of Total Saline Matter.**—(1.) Measure 10 c. c. into a small porcelain crucible; (2.) Evaporate to dryness; (3.) Add about ten drops of nitric acid; (4.) Heat the crucible to dull redness; (5.) Suffer it to cool, and add ten more drops of nitric acid; (6.) Heat it up again gradually to a moderately strong heat, until all the carbon is destroyed and the residue is white; (7.) Let it cool, and weigh.

10. **The Specific Gravity of Urine** is best taken by measuring 100 c. c. into a beaker or flask whose weight is accurately known. The increase of weight in grains will be the specific gravity, water being 1,000. Instead of 100 c. c., 50 or 25 c. c. may be taken, when the weight, multiplied by 2 or 4, will be the specific gravity (SUTTON). The urinometer gives the least accurate of all results.

For further details regarding the processes of volumetric analysis the reader is referred to the excellent little treatise of Mr. Francis Sutton, already noticed.

SECTION VIII.—THE MICROSCOPIC EXAMINATION OF THE URINE, AND THE PATHOLOGICAL RELATIONS OF THE DEPOSITS.

In perfectly healthy urine there ought to be no sediment whatever, unless it be the very merest haze of mucus, or the slightest precipitate of urates caused by a low temperature. Even these may be abnormal (PARKES).

The sediments have been broadly arranged by Dr. Parkes into the following three classes (l. c.):—

CLASS I.—SUBSTANCES SUSPENDED IN THE URINE WHICH HAVE NEVER BEEN DISSOLVED.

They commence to precipitate as soon as the urine is passed. The most important sediments belong to this class, and consist chiefly of organic bodies derived from the structures composing

the urinary organs, or of the *productive* effects of disease upon the kidney, such as *inflammation*, tubercle, cancer. They often afford the only signs of kidney disease, or of the implication of the kidney in some general affection. They are made up of the following substances:—

1. *Mucus and Epithelium from the Urinary Passages.*—In many diseases the quantity of epithelium from the bladder is increased, indicating catarrhal inflammation of the mucous membranes. The epithelial cells are of various sizes and stages of formation (fig. 47); and frequently free nuclei are seen. In catarrh of the bladder the mucus, from its cohesion, is apt to form thin transparent flakes or cylinders, resembling casts from the prostate or kidney.

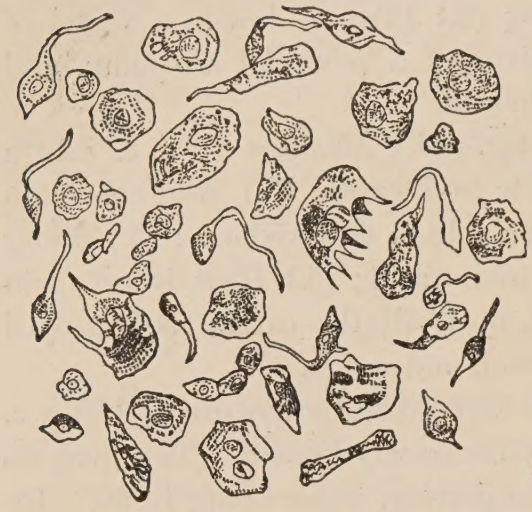


Fig. 47.*

The epithelium-cells from the pelvis of the kidney are often triangular or caudate, with well-defined nuclei. They generally adhere together in groups of three to ten, when they appear to have an imbricated arrangement, and perhaps are more closely connected than natural, by adhesive mucus (fig. 48). They are never found in healthy urine, but are present very commonly in catarrhal and calculous cystitis. Tailed or caudate cells are also sometimes present with pelvic epithelium.

Fig. 48.†

The epithelium from the ureter is columnar in its character (fig. 49), and not unlike that found in the male urethra, which is mostly columnar, and is more flattened than that of the bladder, and less regular than that of the pelvis. Mixed with it there is a good deal of scaly epithelium, especially towards the orifice of the urethra.

Fig. 49.‡

Large cells of scaly epithelium are also often met with in

* Epithelium from the bladder. Many of the large cells lie upon the summit of the columnar and caudate cells, and depressions are seen on their under surface. One is seen near the centre of the figure (after Dr. Beale, *On the Urine*, p. 259).

† Epithelium from the pelvis of the kidney (after Dr. Beale, l. c., p. 188).

‡ Epithelium from the ureter, (after Dr. Beale, l. c.)

the urine of females. They are derived from the vagina. They vary much in size and form, and are sometimes very irregular in shape, with uneven ragged edges (fig. 50).

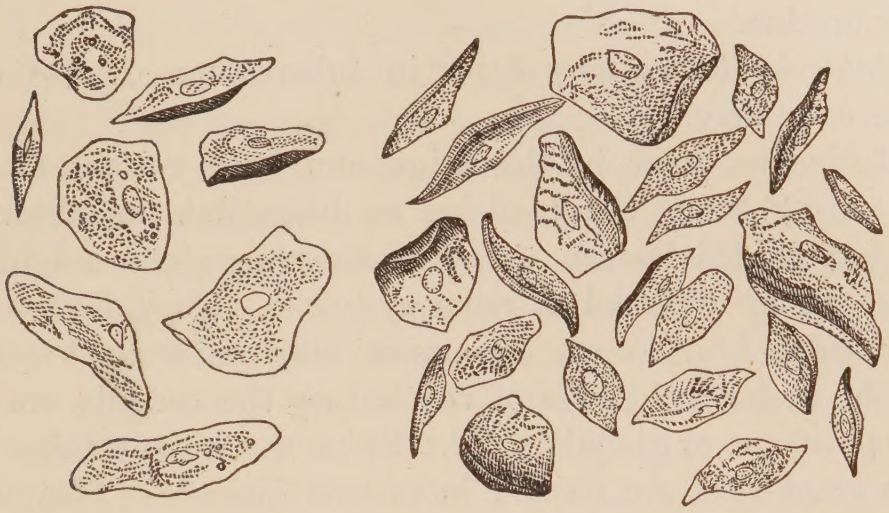


Fig. 50.*

Renal epithelium is only found in disease. It consists of round or slightly compressed cells, or masses of material, with well-defined central portions or nuclei, which are not cleft like the pus nucleus under the action of acetic acid, but become at first more defined and afterwards paler and smaller. In the urine they are less polygonal and more rounded than they are in the renal canals (fig. 51). Their presence indicates more or less desquamation from the tubes; while their morbid condition and admixture with other products may indicate still greater disease. Thus it is sometimes fatty; the whole space between the nucleus and the cell-wall being filled with fatty globules; but these changes and their value have been fully considered under chronic Bright's disease, p. 123, *et seq.*

Fig. 51.†

2. *Other Cell-Forms occur in the Urine, the productive results of inflammatory diseases* of the mucous membrane of the urinary passages. They are chiefly granules, singly or in clusters, and pus. The pus is known by its nucleus becoming cleft into two, three, or five divisions, under the action of acetic acid. The amount of pus is generally far greater in *cystitis* than it is in *pyelitis* or

* Vaginal epithelium from urine (after Dr. Beale, l. c., p. 260).

† Epithelium from convoluted portion of uriniferous tube.—(*a.*) Treated with acetic acid × 215 (after Dr. Beale, l. c., p. 187).

nephritis; and wherever there is a markedly large quantity of pus it is generally from the bladder, unless there be a prostatic or other abscess communicating with the urethra.

3. *Cancer-Cells* occur in cancerous disease of the bladder, kidney, or urethra.

4. *Tubercle-Masses* may occur in tuberculous disease of the bladder or kidney.

5. *Cylinders* occur in the urine, and have various modes of origin:—(*a.*) From the *bladder,* as long, flat, membraniform, twisted, or folded bodies. (*b.*) from the *prostate,* as coagula, two or three times as broad as renal cylinders. They are soluble in acetic acid. Amylaceous corpuscles may exist in them. (*c.*) From the *ureter* and *pelvis* of the kidney the coagula are cylindrical, pyriform, or globular. (*d.*) From the kidney-tubes cylinders or renal casts are formed in various diseases. They vary in breadth from $\frac{1}{3000}$th to $\frac{1}{10000}$th of an inch—*i. e.,* from about the breadth of the straight renal tubes to half or a third of that size. Their length varies from $\frac{1}{2000}$th to $\frac{1}{300}$th of an inch. The terms used by Dr. George Johnson, to whose researches on diseases of the kidney we owe much, express very well the special character of the several varieties of casts. They are—(1.) Epithelial casts; (2.) Large waxy casts; (3.) Small waxy casts—the hyaline casts of Vogel and Basham; (4.) Granular casts; (5.) Oily casts; (6.) Bloody casts; (7.) Purulent casts.

Solution of iodine is the best re-agent for making the waxy hyaline or transparent casts visible. The chemical composition of these casts is unknown. They are probably albuminous.

6. *Kidney-Structures* may occur in the urine if the structure of the kidney has commenced to break up.

7. *Blood-Corpuscles* indicate ruptures of vessels somewhere in the urinary passages. Copious hæmorrhage is usually from the bladder, carcinoma of the kidney, or calculus of the pelvis. The corpuscles are often very much modified in form.

8. *Fibrin* is present in many cases of bloody urine.

9. *Corpora amylacea* may occur from the prostate gland.

10. Fat. 11. A fatty substance, to which the name of urostealith has been given. 12. *Spermatozoa.* 13. *Sarcinæ.* 14. Hair. 15. *Various entozoa* have been occasionally found in the urine. (See under "Entozoa.")

CLASS II.—SEDIMENTS FORMING IN THE URINE AFTER SECRETION, BUT WHICH MAY DEPOSIT IN THE RENAL PASSAGES OR AFTER EMISSION, EITHER IN CONSEQUENCE OF CHEMICAL CHANGES OR FROM CHANGE OF TEMPERATURE.

1. *Uric Acid* in various combinations with bases, such as *soda, potash, lime,* or *ammonia,* and more or less coloured with urine-pigment. They are associated sometimes with phosphate or oxalate of lime. The uric acid sediments in combination with bases are generally associated with increased acidity of the urine, either by normal acids, such as uric, sulphuric, phosphoric, hippuric, or with the formation of acids developed after emission. They are known as "urates," and commonly spoken of as yellow lateritious "brick dust," or "fever sediments." They dissolve when the urine is heated to 130° Fahr. They are also soluble in potash and liquor ammonia. Acids decompose them after warmth and liberate uric acid. Three forms are distinguishable—(1. Irregularly formed or amorphous particles; (2.) Round globules of various sizes; (3.) Fine acicular prismatic crystals. These forms may deposit at *two* different periods after emission—(1.) As soon as the urine has cooled down to the temperature of the atmosphere. In such cases the water is diminished—the urine being that known as febrile urine; or it occurs when there is an absolute increase of uric acid. (2.) They deposit some hours after the urine has been passed, and long after the urine has been of equal temperature with the air. Under these circumstances the deposit betokens increased acidity from changes in the pigment or extractives, or uroxanthin. The acids so formed may be lactic, acetic, or butyric; and a drop of acid added to such urine will anticipate the deposit.

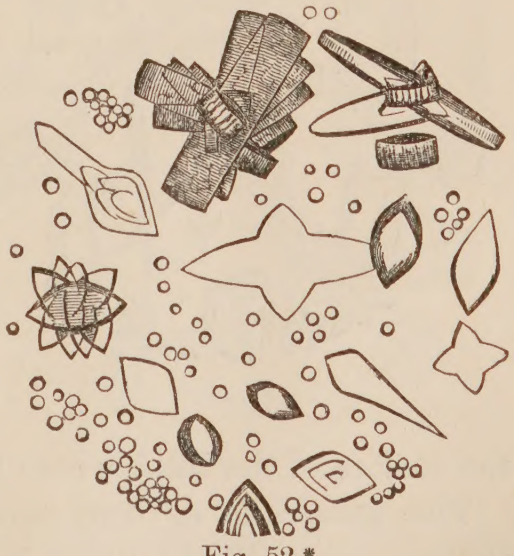


Fig. 52.*

2. *Uric Acid Sediments* in their pure state occur in the form of rhombic prisms, or rhombic plates, or of thin hexagonal plates like cystin; but the most usual forms are referable to some

*The most usual forms of uric acid sediment, with blood-corpuscles intermixed (after Dr. Otto Funke).

variety of the rhomb (fig. 52). Such deposits often present the aspect of a granular sand of a golden lustre, sometimes mingled with blood-discs; and generally the deeper the colour of the urine the darker is the uric acid sediment. Its appearance does not necessarily indicate that an excess of uric acid is forming in the body. The urine is generally yellow and transparent, and the acid is deposited slowly without admixture of urates. Liquor potassæ, and also nitric acid in excess, dissolve uric acid.

3. *Sediments of Hippuric Acid.*—Owing to the solubility of this acid its sediments are rare. When it does occur, it is in the shape of long four-sided, acuminated prisms, or acicular needles fixed on uric acid crystals, with which they are sometimes confounded, as well as with phosphates. They are distinguished from phosphates by being insoluble in acids; and from uric acid they may be separated by boiling with strong alcohol (PARKES).

4. *Sediments containing Phosphoric Acid* are formed of the *ammoniaco-magnesian phosphate*, the *phosphate of lime*, and the *phosphate of magnesia.* The ammoniaco-magnesian phosphate occurs in the form of beautiful transparent prisms, or in foliaceous, penniform, or stellar prisms or crystals.

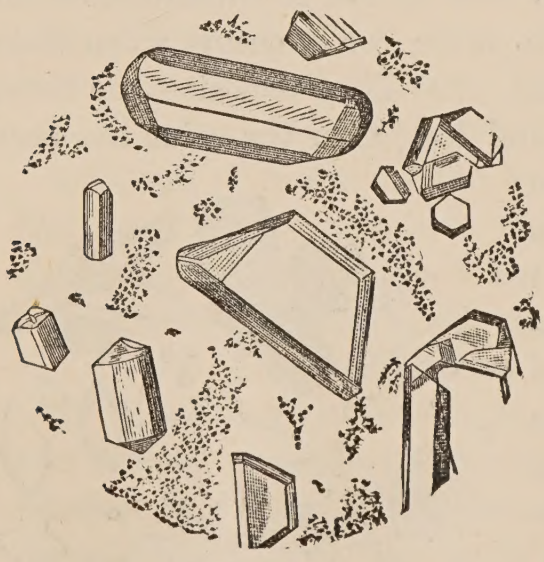


Fig. 53.*

The *phosphate of lime* and the *phosphate of magnesia* form a white amorphous powder, or they occur as round small globules, or as prismatic crystals with oblique summits (HASSALL).

Acids dissolve all those sediments; heat has no effect upon them.

The *ammoniaco-magnesian phosphate* is almost always deposited as a result of the decomposition of urea, and the deposit usually commences on the surface of the fluid, where the urea is most exposed to the air.

The *phosphate of lime* and the *phosphate of ammonia* are thrown down if the urine becomes alkaline from fixed alkali; as after vegetable food or the carbonates of the fixed alkali, or

* Ammoniaco-magnesian phosphate in prisms, mixed with amorphous granules, phosphate of lime, and granular urates (after DR. THUDICHUM).

after the salts which form them have been taken. Decomposition of the urea in these cases is generally rapid, and *ammoniaco-magnesian* phosphate is also produced. Such, also, is the case with urine of chronic diseases, or during convalescence from acute diseases, when the urine is feebly acid. The phosphates and urates occur together under the following conditions:—(1.) When urates having been deposited, the urea decomposes to a slight extent—enough to form the ammoniaco-phosphate, and yet not enough to dissolve the urate; (2.) When crystallized uric acid has formed and been acted upon by the ammonia formed from the urea, the crystals of uric acid disappear, and are replaced by round globules of *urate of ammonia*, mixed with the precipitate of phosphates. These deposits are always white, and unchanged by heat; soluble in dilute hydrochloric acid, but insoluble in ammonia and in liquor potassæ. Mucus, pus, or blood may mask the chemical re-actions. A small quantity of solution of sesquicarbonate of ammonia, added to a large quantity of healthy urine, will yield prisms of the triple phosphate.

5. *Sediments composed of Oxalate of Lime.*—Opinion is very undecided as to the significance of this deposit. It occurs in one out of every three cases, taking the cases indifferently as they occur in an hospital. It appears (fig. 54) in four forms;—(1.) As octahedra (*a*); (2.) As hour-glass, contracted, or dumb-bell-like bodies (*b*); and (3.) Compound octahedra (*c*) may also be seen; (4.) As small, flattened, bright discs, very readily mistaken for blood-discs.

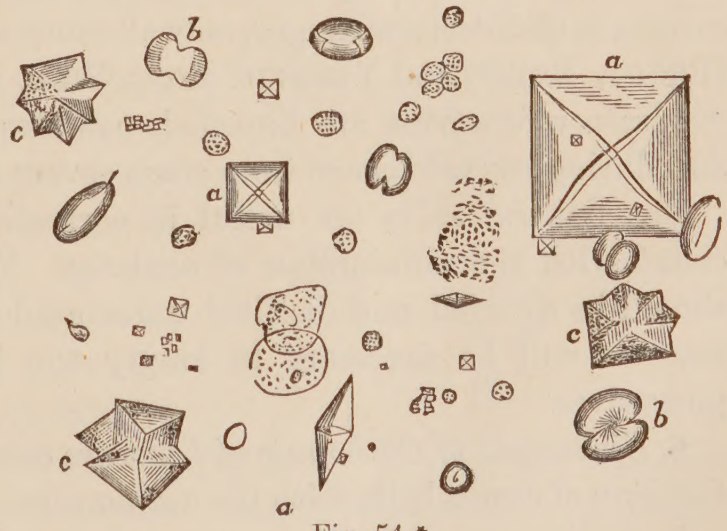


Fig. 54.*

Oxalate of lime, although found in the blood, is probably a result of chemical changes in the renal passages. The octahedra grow or increase in urine after it is passed; but the dumb-bell crystals are said not to do so. Dr.

* (*a.*) Octahedra; (*b.*) Dumb-bell; (*c.*) Compound octahedra; circular and oval crystals of oxalate of lime, (after Dr. Beale, l. c.)

Beale considers that the dumb-bell crystals of oxalate of lime are mainly formed in the kidney-tubes. He found them most frequently in casts. Dr. Parkes considers that the oxalic acid of urine most probably results from uric acid; and may be a substitution for the excretion of carbonic acid of the lungs (SCHMIDT). In order that oxalic acid shall form in the urine, there must either be irritation from a calculus, or from some other cause, or there must be fermentive changes from mucus. If there be no evidence of any of these conditions, the oxalic acid of fresh urine may be presumed to come from the blood (PARKES). Crystals of oxalate of lime are insoluble in water—are unaltered by boiling either in *acetic acid* or in *liquor potassæ;* and are soluble in nitric acid without effervescence.

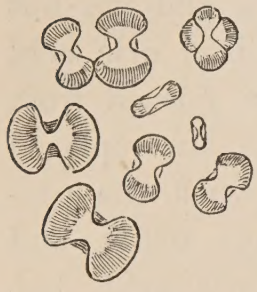


Fig. 55.*

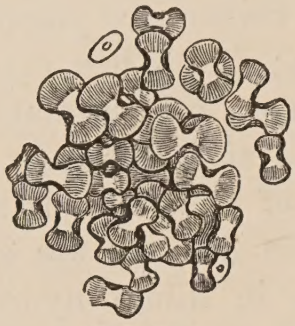


Fig. 56.†

Pathological Relations.—Many articles of diet contain oxalic acid in abundance; but there are also certain articles of food and drink the use of which, when digestion is deranged, is followed by the formation of oxalate of lime and its presence in the urine. Sugar in excess, the use of frothy sparkling beer or wine, turnips, parsnips, carrots, cauliflower, asparagus, may all cause a "temporary oxaluria" (PROUT, ROBIN, and VERDEIL, ROSE, BIRD, HASSALL). When the respiratory functions are impeded, oxalates appear in the urine, and during convalescence from some severe diseases, as *typhus.*

The mineral acids are useful in correcting the dyspepsia associated with the elimination of oxalates. Water containing lime should be avoided, and distilled water used instead; and in many cases it will be necessary to empty the intestines by suitable purgatives.

6. *Sediments of Carbonate of Lime* are occasionally associated, in the form of dumb-bells, with the *ammoniaco-magnesian phosphate.*

7. *Sediments of Leucine* are precipitated in round corpuscles, sometimes with a concentric form, and look like heaps of fat; but

* Dumb-bell crystals of oxalate of lime, of very regular form, from the urine of a child two years of age, suffering from jaundice (after DR. BEALE, l. c., p. 300).

† Collection of dumb-bells, such as often forms the nucleus of a calculus (after DR. BEALE, l. c., p. 300).

when crystallized from pure solutions, it appears as fine, dark-coloured needle-like crystals. To recognize it fully, it must be separated by careful sublimation (PARKES). The suspected leucine is to be placed on platinum, carefully moistened, and then dried with nitric acid. The almost imperceptible flake which is left is to be moistened with caustic soda, and evaporated carefully over a spirit-lamp. If leucine is present, it forms an oily-looking drop (SCHERER).

Pathological Relations.—It is believed to be the result of the disintegration of certain of the nitrogenous tissues of the principal glands, and occurs particularly in *acute yellow atrophy* of the liver, where it is found in the substance of the liver itself.

8. *Sediments of Tyrosine* are of a greenish-yellow colour, composed of heaps of fine needles, which can be obtained on evaporation. It ought to be treated with nitric acid, like *leucine*, and then a little *liquor soda* used. The nitric acid gives a deep orange-yellow colour, which becomes deep yellow on evaporation. The soda gives the yellow flake a red tinge; and on heat and evaporation, a black-brown residue is left (SCHERER, quoted by PARKES).

Pathological Relations.—It is found wherever leucine is met with.

9. *Sediments of Cystin* form a white or light fawn-coloured, amorphous, rather bulky precipitate, or they appear at once as six-sided plates (fig. 57). In both cases ammonia dissolves it,—so do fixed alkalies and their carbonates; and from this solution it crystallizes on spontaneous evaporation. It does not disappear when the urine is gently warmed; and it is insoluble in carbonate of ammonia, in dilute hydrochloric acid, and in acetic acid.

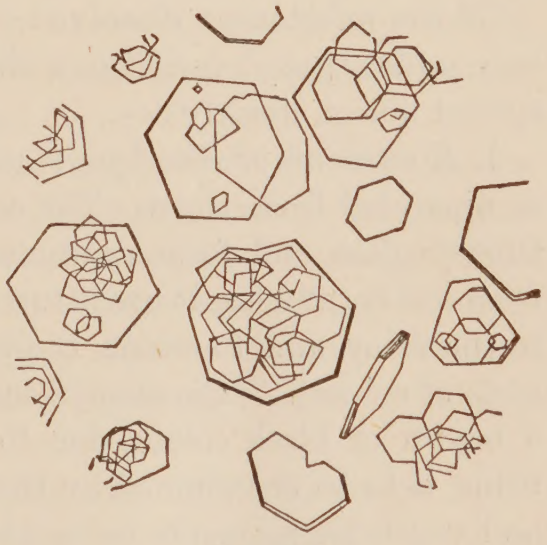


Fig. 57.*

Pathological Relations.—It is associated with the excessive elimination of *sulphur*. Its persistence in the urine is often hereditary, and is generally associated with derangement of the

* Cystin precipitated by acetic acid from its ammoniacal solution (after THUDICHUM).

functions, or organic disease of the liver. It occurs also in cases of chlorosis.

CLASS III.—SEDIMENTS COMPOSED OF SUBSTANCES FOREIGN TO THE URINE, AND WHICH ACCUMULATE IN THE URINE ALWAYS AFTER EXPOSURE TO THE ATMOSPHERE.

1. *Fungi* in acid urine—*e. g.*, the *Penicillum glaucum*, its *spores*, *thullus*, and *fructification*.

2. The *Torulæ cerevisæ* in saccharine urine begins to form in two or three hours after emission. It forms a gelatinous mass, composed of *sporules*, which subsequently develop into *beaded threads;* and in a few days aërial fructification appears.

3. *Vibriones* and *Monads* occur in urine containing much mucus, and which is feebly acid or alkaline.

All urine should be collected in glass vessels, which are scrupulously clean; and *the quantity operated upon should embrace the whole urine passed during the twenty-four hours.* All sediments should be examined—(1.) Within a few hours after excretion; (2.) After twenty-four hours.

Of the substances dissolved in the urine, or becoming obvious only under special conditions, there are some which require more special notice, namely:—

1. *Hæmatine, or Blood-pigment*, occurs either as blood-corpuscles or separated from them. The corpuscles fall to the bottom of the tube or glass, and form a sediment. If the hæmatine is separate from the corpuscles, it constitutes a pigment completely dissolved in the urine, and to which it gives a brown or black colour (see scale of colour). It is always accompanied by albumen. Urine of a brown or black colour, due to blood-pigment dissolved in the urine, is to be determined by the elimination process in part—bile and vegetable pigments being absent.

Urine containing blood in obvious quantity permits it generally to coagulate into blackish masses like pieces of black currant jelly; and when it partly coagulates in the bladder, linear masses of clot, of nearly the shape of leeches, are passed from the urethra. The urine has a port-wine hue, and abundance of entire red discs of blood-corpuscles may be detected with the microscope. These sink with readiness to the bottom of the vessel. Their non-granular surface, uniform size, and yellow colour, are characters sufficient to identify them.

Blood-pigment, or hæmatine, dissolved in the urine, does not necessarily indicate local disease or rupture of vessels in part of the urinary organs. It must be regarded as indicating rather a specially morbid condition of the blood, as is associated with septic poisons, or with profound cachectic diseases (PARKES). It may be observed in *typhus fever, malignant variola, remittent fever, yellow fever, scurvy,* and *Bright's disease.* Albumen is said always to co-exist.

2. *Albumen.*—Its presence must be regarded as an indication of disease, although it has been observed to occur in small quantity, and temporarily, in the urine of some persons supposed to be at the time in a healthy state; although it is more than probable that slight disease is present, or that such grave affections as Bright's disease may be commencing, to terminate fatally perhaps two years later (Christison, *Monthly Journal,* Jan., 1851; PARKES, p. 184). Very slight disease—not more than what appears to be dyspepsia—may have albuminuria associated with it, of a temporary nature. It has been observed in *intermittent fever, typhoid fever, rubeola, variola, pneumonia, pleuro-pneumonia, scarlatina, bronchitis, pleurisy, peritonitis, intestinal catarrh, follicular enteritis, acute rheumatism, chlorosis, cardiac diseases, phthisis* (SOLON, FINGER, PARKES).

Hæmaturia has been found associated with albuminuria in *scarlatina, rheumatism, arthritis, typhus* (during the second week), *nephritis (calculous?), pneumonia, erysipelas, torpor, hepatitis, peritonitis, phthisis, chlorosis, intense oxaluria, phosphatic diathesis, last month of pregnancy, uterine disease, bladder-cancer, internal use of turpentine* (Heywood Thomson, *Lancet,* July, 1857; PARKES).

Dr. Parkes desires to draw a strong distinction between "temporary" and "permanent" albuminuria. The former ought to imply that the albuminuria, after lasting for some days, or even weeks, finally disappears entirely. The latter implies that albuminuria does not disappear. Dr. Parkes found temporary albuminuria, with the quantity of albumen large, in acute lobar pneumonic cases and acute renal cases, such as Bright's disease. He found albumen considerable in amount in *typhoid, variolous,* and *scarlatinal cases,* and small in amount in *paraplegia (spinal), hemiplegia, chronic phthisis, acute pleurisy, acute bronchitis, in hypertrophy, dilatation, and valvular affections of the heart, in*

acute and subacute rheumatism, in purpura hæmorrhagica, in typhoid and typhus fever, and in erysipelas (PARKES, p. 188).

"Permanent albuminuria" he found associated with all forms of *Bright's disease,* with *encephaloid and cystic disease of the kidney,* with *leucocythæmia and presumed lardaceous disease of the kidney, in chronic phthisis, and in pleurisy, where casts and kidney-structures were seen in the urine, in hypertrophy and valvular affections of the heart, in cerebral softening of hemiplegia, in pancreatic disease, in purpura hæmorrhagica, and in typhoid fever.* The result is, that if heart diseases be excluded, "permanent albuminuria" indicates kidney disease invariably, and the distinction between "temporary" and "permanent" albuminuria is apt to be drawn with insufficient care; and in all cases where it occurs there is either congestion and increased lateral pressure on the vessels of the kidney, or there is absolute structural disease of their walls. In the Mauritius, and in Bourbon, a singular hæmaturia sometimes occurs, in which a varicose condition of the lymphatics has been supposed to cause the albumen (PARKES, p 190).

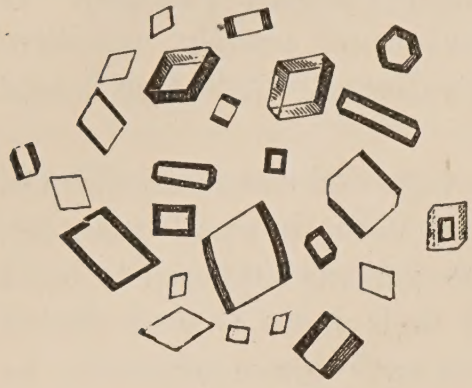


Fig. 58.*

3. *Urea.*—It is often of importance to determine the mere presence of urea in a small amount of fluid, as in the fluid of the

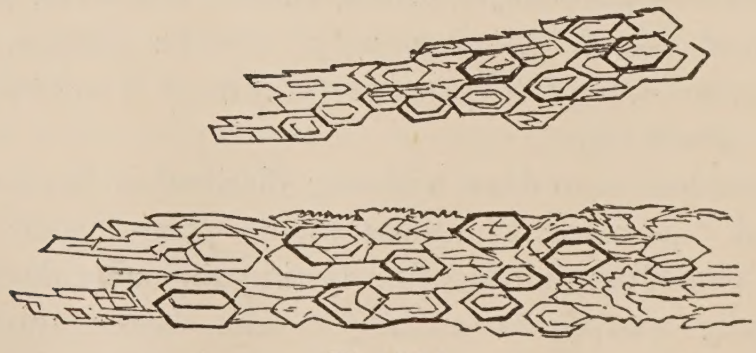


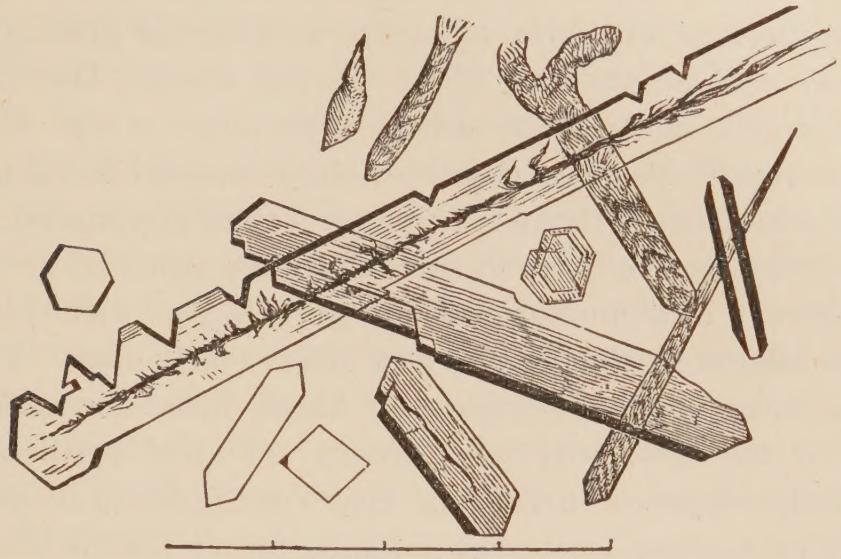
Fig. 59.†

ventricles of the brain. The process has been already explained at p. 267; and if, in place of nitric acid, oxalic acid is used, we obtain crystals of *oxalate of urea* (figs. 58, 59).

* Oxalate of urea—perfect crystals (after BEALE). "Urinary Deposits," No. II., Plate IV.

† Oxalate of urea (after BEALE). "Urinary Deposits," No. II., Plate IV.

The alcoholic extract of urea leaves, on spontaneous evaporation, acicular crystals of the following form (fig. 60):—



100th of an inch × 42.

Fig. 60.*

Section IX.—Detailed Description of Diseases of the Kidney.

ACUTE BRIGHT'S DISEASE—Syn., ACUTE ALBUMINURIA; ACUTE DESQUAMATIVE NEPHRITIS; ACUTE RENAL DROPSY.

Latin Eq., *Morbus Brightii acutus;* French Eq., *Maladie de Bright aiguë;* German Eq., *Acute Bright'sche Krankheit;* Italian Eq., *Malattia del Bright acuta.*

Definition.—*An intense febrile disease, which may come on after scarlatina, and other exanthemata, or independent of these, and which is marked by signs of intense congestion of the kidney, with exudation and hæmorrhage into the tubes, and desquamation of the epithelium. The secondary phenomena are uræmic symptoms to a greater or less degree, and in the majority of cases general dropsy* (Frerichs, Parkes, Johnson, Rayer).

Pathology.—This affection seems to bear a similar relation to chronic Bright's disease that cases of acute phthisis bear to scrofula. It is described under the name of "*acute desquamative nephritis*" by Dr. George Johnson (whose investigations have especially elucidated this form of kidney diseases); and corre-

* Oxalate of urea from urine—extraction by alcohol, and an oxalate formed by the addition of oxalic acid.

sponds with the acute inflammatory dropsy of many writers. Elements closely allied to pus form in the kidney-tubes.

In the rapid forms of Bright's disease the products in the urine occur at first in the form of casts—a catarrhal process. These casts may accumulate and block up the tubes. The kidney is then enlarged, of a white colour (the large white kidney of Bright). Acute dropsy is constant, often ascribed to cold, or as a result of scarlatina. Urine may be for a time suppressed, and the little which passes is of a red-brown colour, generally from blood. The sediment is abundant and deeply tinged with blood. It contains albumen, blood-casts, and renal epithelium. The sediment is "composed of coagulated fibrin, blood-corpuscles, cells having for the most part the character of renal epithelium, and occasionally crystals of uric acid. Some of the fibrin is coagulated in irregular masses, having no definite form; this is always the case when the hæmorrhage has been abundant and rapid, so that much of the blood has escaped from the kidneys before it has had time to coagulate; but with these masses there will be seen numerous cylindrical bodies composed of fibrin, which, having exuded from the Malpighian bodies, have coagulated in the tubes, and, escaping thence, present solid cylindrical moulds of the interior of the tubes, in which are entangled blood-corpuscles and epithelial cells, which have been shed by a process of desquamation from the surface of the tubes" (Johnson, *On the Kidney*, p. 89). To such casts, characterized by the presence of recently formed and entire epithelial cells, Dr. Johnson proposes the name of "*epithelial casts*"—their average diameter being about $\frac{1}{700}$th of an inch. Death, sometimes after only a fortnight's illness, discloses a large soft kidney, with swollen cortical substance of a dark colour, on removal of the capsules, and the surface much injected. It exudes, on section, drops of blood. To the microscope the tubes are opaque, and are filled with lymph-corpuscles and granular matter. Some have lost their epithelium, and are filled with coagulated fibrin, which, if expelled, would form a fibrinous cast.

At a later period the pyramidal portions are still of a dark colour; but afterwards the cortical part becomes paler and softer, the surface not smooth when cut, and giving out a turbid fluid when squeezed. The tubes are seen to be denuded of epithelium, and filled with masses of secretion, which take on the forms of the tubes. Scanty and bloody urine are condi-

tions associated with engorgement of the Malpighian tufts. These are visible as red specks to the naked eye, and the tubes may be found filled with blood, or containing hæmatine. The morbid process continuing, secretion collects in the tubes and fills them to an extreme degree, and the more it accumulates, the larger and whiter the kidney becomes. The material can be seen with the naked eye in the cortical part. The surface is pale and smooth, with stellate or arborescent venation, surrounding numbers of opaque white spots. A section shows similar appearances, but the white material is now seen disposed in lines. If a congeries of tubes are swollen between the blood-vessels, granulations are thus formed. The new material consists of large masses of dark, granular, and fatty matter, which distend the tubes.

A section shows, microscopically, large portions of tubes quite opaque; their natural lining is gone, and contiguous tubes become united or massed together by the new material which fills them; and it is often difficult to determine whether the partition-membranes of the tubes exist or not (WILKS).

Granular matter may also be seen in some parts of the straight tubes. It is exceptional to find this material in the capsule of the Malpighian tufts.

The *Urine* in the early stages and height of the disease presents intensely febrile characters. It is small in quantity, deeply pigmented, and deposits urates. It contains a variable but usually a large amount of albumen and blood. The sediments consist of desquamated kidney, ureter, and bladder-structures, and voided renal cylinders, and sometimes large masses of coagulated fibrin, or partly decolorized clots. Urea is augmented; and when it appears to be below the normal amount, its exit is impeded, and uræmic symptoms are present, which generally increase and prove fatal. When the urine is very scanty, it becomes almost solid by heat. When the disease is about to end fatally, the quantity of water and of the solids decrease, the diminution of the solids being more considerable than that of the water. The albumen decreases least, and is very abundant till the last. When recovery is about to take place, *diuresis* usually occurs; and often an enormous quantity of water is passed, containing much *urea* and *chloride of sodium*. The albumen at the same time diminishes and disappears, and the kidneys recover perfect health (PARKES, l. c., p. 378).

Causes.—The poisons of *scarlatina, cholera, measles,* and *erysipelas;* intemperance in alcoholic drinks; the exhausting influence of previous disease; deficiency of food, with fatigue and mental anxiety; exposure to cold and wet.

Treatment.—(1.) Relieve the kidneys as much as possible from the labour of elimination, by avoiding exposure to cold, by keeping the patient at rest in bed, in a room of moderate uniform temperature. (2.) The food should be scanty, consisting of gruel, arrow-root, milk, or weak broth; pure water is the best drink, and alcoholic fluids are not to be taken on any account. (3.) Free action of the skin and bowels must be maintained. The *hot air bath* and antimonial remedies are the best agents to effect the first of these conditions, and free perspiration is to be encouraged by bedding the patient in blankets. *Antimonial wine* may be given in doses of from fifteen to thirty drops every four or five hours. The bowels are to be kept open by the *compound jalap powder,* in doses of twenty to sixty grains, repeated daily or on alternate days. It may be alternated with *podophyllin,* or with extract of *colocynth.* Mercury is not to be given. (4.) Cupping over the loins relieves pain in the back, and the quantity of urine passed generally increases after eight or ten ounces of blood have been withdrawn in this way from an adult, or two or three ounces from a child three or four years old. (5.) When the tongue becomes clean and the general symptoms improve, mutton broth or good beef-tea may be indulged in; and, as the digestion improves, solid food may be eaten in small quantities, beginning with fish and fowl, and afterwards mutton or beef. (6.) Flannel must be worn next the skin. (7.) Iron is of great service during convalescence, for in such cases the anæmia becomes extreme. Phosphate of iron in the form of *syrup,* or *citrate* of *iron* and *quinia,* or the *ferrum reductum,* are the most digestible forms, and they ought to be given in small doses repeated after every diet. 8. Diuretics are not to be given (see JOHNSON, *loc. cit.,* p. 125 to 138). In a case where the urine was suppressed, fomentations consisting of infusion of the leaves of digitalis were found by Professors Christison and Vogel to increase enormously the amount of urine. Dr. Parkes found the amount of the albumen to diminish markedly from the use of the tincture of the sesquichloride of iron (l. c., p. 379).

SUPPURATIVE NEPHRITIS.

LATIN EQ., *Nephritis suppurans;* FRENCH EQ., *Néphrite suppurée;* GERMAN EQ., *Interstitielle Nephritis;* ITALIAN EQ., *Nefritide suppurativa.*

Definition.—"*Inflammation, with suppuration of the substance of the kidney.*"

Pathology.—The general nature of inflammation having been fully considered in volume first, it is of importance, in studying the inflammations of the kidney, to determine—(1.) The tissue or tissues affected—vessels, parenchyma, or interstitial connective-tissue; (2.) Whether only the cortical substance, or the pyramidal, or the pelvis of the kidney, is affected, or all parts together; (3.) Whether the affection is partial or diffused.

In the commencement of the disease a definite tissue can generally be indicated by microscopic examination after death, as the starting-point of the affection; but later it is not so easy. The several tissues are often consecutively attacked.

There are two essentially different affections,—(1.) Parenchymatous nephritis; (2.) Interstitial nephritis; and these may be complicated with the fatty or the amyloid degenerations about to be noticed. All forms of disease may be present at once, sometimes one and sometimes another occurring first; but *parenchymatous nephritis* is most generally the primary affection (Rasmussen, *Med.-Chir. Review*, July, 1863).

The cells of the convoluted tubes appear to be larger and richer in albumen than those in the straight ones; and any disease of the former, rendering them inactive, is of far more serious import than disease of the latter, producing an actual change in the urine. Affections of the latter portion of the kidney only—namely, the pyramidal—are usually of a catarrhal nature, and are described by Rasmussen as "*papillary catarrh*," or "*catarrhal nephritis.*" The catarrh affects principally the straight canals and papillæ, and is often continued from the bladder and urethra. Its exciting causes are stimulant, alcoholic, or terebinthine drinks, the use of cantharides, or of acid diuretics. It may be the starting-point of parenchymatous nephritis, and often complicates it. Such cases are generally suppurative, especially when secondary to bladder inflammation, or paralysis of the bladder in spinal affections. The suppuration is then gener-

ally most obvious and active in the cortical portion of the organ. The kidney is enlarged, and its surface covered with minute points of pus or small abscesses. A section of the whole kidney shows that these abscesses are diffused throughout the cortical, to the exclusion of the pyramidal part. The mucous membrane of the pelvis is inflamed, and is described under the name of pyelitis (WILKS). When the capsule is highly vascular and involved in the process, it indicates a chronic affection, or is a result of a former inflammation. In cases of uncomplicated catarrhal nephritis, where suppuration does not occur in the cortical portion, the lesions are confined to the canals and papillæ. They present a whitish or yellowish striation, with hyperæmia of the intervening vessels, and bloody ecchymosis may be present over the whole kidney, especially if acid diuretics have been largely given. If the disease continues long, the distended urinary canals press on the blood-vessels, when the hyperæmia generally ceases. The productive effects of the inflammation are limited to cell-growth (nucleated, club-shaped, fusiform, or ramifying), mixed with mucous catarrhal products (mucin), sometimes combined with fatty metamorphosis or other degeneration of epithelium.

True parenchymatous nephritis is a hypertrophy or "cloudy swelling" (see p. 124) of the large cells of the convoluted tubes. The cells take up large quantities of the *albuminates*, becoming distended, turbid, granular, and closely adherent to each other. Subsequently all the cell-forms vanish, and a large granular fatty mass is set free, which generally forms into "inflammatory globules." It often co-exists with "interstitial nephritis." When catarrh exists, it is usually associated with the symptomatic phenomena of Bright's disease. Retardation of venous blood in the kidney is a common event in parenchymatous nephritis. Thrombi are then apt to form in the veins of the kidney, and being transported to the *vena cava* and heart, may find their way into the lungs. In the advanced stage of the disease, resolution or recovery corresponds to fatty degeneration of the cells, sometimes with loss of substance apparent on the surface, indurations, granulations, cysts, in connection with or separated from the urinary canals. The interspaces between the granular elevations consist of empty collapsed tubes. The granular cirrhotic appearance of the kidney is not similar in pathological origin to that of

the liver. In the liver it is the inter-acinous connective-tissue which is first affected, and causes the cirrhotic granulations; in the kidney, on the contrary, the parenchymatous cells of the tubes become first affected, and the lesion is afterwards complicated with *interstitial nephritis*. The Malpighian glanduli ultimately become small, corrugated, and surrounded by thickened capsules of connective-tissue. These capsules, with the epithelium, sometimes eventually become fatty, amyloid, or calcareous. With regard to *interstitial nephritis*, it has been doubted whether there is a connective-tissue or fibrous matrix to the kidney. I have never yet seen a human kidney in which it is not demonstrable. It becomes immensely hypertrophied, and minute cells grow up in its substance in large numbers, even to the extent of suppuration. Atrophy is also a usual result, first of all at the expense of the cortical part. On section it is seen to be diminished between the pyramids and the surface. The edge is irregular, and the surface is granular. The granulations are composed of bundles of tubes lying between veins, and are not to be confounded with white specks of deposit. The capsule separates with great difficulty, tearing the tissue which adheres to it. The glomeruli appear to be much closer together than natural (Dickinson, *Med.-Chir. Trans.*, 1863). Cysts are frequently met with on the surface.

Sometimes the whole of the new interstitial material undergoes the fatty degeneration. Granulations are also formed by the connective-tissue contracting round the canals and glomeruli, and circulation is more or less obstructed. The tubes become constricted in a bead-like manner, and the *tunica propria* is thickened and streaked. The glomeruli become small, homogeneous, and more or less fatty.

Circumscribed interstitial nephritis often affects the pyramids only, as a result of syphilis; nodes are the consequence, which, passing away or diminishing, leave cicatrix-like depressions not unlike those from hæmorrhagic infarctions (Rasmussen, Wilks, Virchow, Dickinson).

The kidneys may suppurate—(1.) From morbid conditions of the blood; (2.) From external violence; (3.) From retention of urine; (4.) From calculi in the kidney (Johnson, *op cit.*, p. 417).

Symptoms.—Uncomplicated acute nephritis has hitherto been considered so rare that its symptoms have not been well de-

scribed. Those mentioned by Baillie are as follow:—"When the kidneys," he says, "are inflamed, more or less pain is felt in the region of these glands, and the pain commonly shoots along the ureters. There is a sense of numbness down the thigh, and in the male there is often retraction of the testicle, or a feeling of pain in it. When one kidney is affected, these symptoms are only felt on that side. The urine is voided frequently, and is sometimes of a pale, but more commonly of a deep-red colour. There is sickness and vomiting. The bowels are at the same time often costive, and subject to collicky pains. These symptoms are accompanied by more or less fever." "When pus is formed, the event may be known by the pus being mixed with the urine." Cases related by Mr. Stanley, however, by no means bear out this description. He gives the case of a man who had retention of urine in consequence of a gonorrhœal discharge being stopped by injections. In this instance the kidneys were found extremely vascular and soft, with numerous minute depositions of pus throughout the cortical and tubular parts, and the infundibula and the pelvis were likewise filled with pus. The principal symptom was severe pain at the fifth lumbar vertebra. In another similar case, but not quite so acute, the kidneys were found so dark coloured as to be almost black, and at the same time remarkably flaccid. This patient died paraplegic, the loss of motion being complete, and that of sensation nearly so.

If nephritis passes to a chronic suppurative state, the pain in the loins is often severe and the appetite impaired, while pus is found often to a considerable amount in the urine, and if a calculus or gravel be the immediate cause, the urine may contain large portions of those substances mixed with blood. Dr. Johnson has clearly shown that the desquamative and suppurative processes in the kidney are much more closely allied than is generally supposed. They pass into each other by almost imperceptible gradations; the products appearing in the urine as purulent casts of the tubes.

Prognosis.—Acute affections of the kidney are in all cases of grave prognosis. The chronic forms of these affections are perhaps consistent with life, but in every case they greatly impair it, and are ultimately the cause of premature death. When pus-corpuscles take the place of renal epithelium the prognosis must always be unfavourable; and the suppurative

process in the kidney is the most rapidly destructive of the lesions. Dr. Johnson has not met with a case of suppurative nephritis which has terminated in recovery (*op. cit.*, p. 440).

Treatment.—The treatment of acute nephritis must be according to the ordinary principles of the treatment of inflammation, by evacuants and opiates. Blisters in such cases are dangerous, and ought to be avoided. The neutral salts, with opiates, are admissible in some cases. Castor oil or other purgative substances which do not act so immediately on the kidneys are more useful, such as compound jalap powder or podophyllin. The general plan of treatment is similar to that which has been already stated.

LARDACEOUS KIDNEY—Syn., AMYLOID DISEASE; WAXY DISEASE.

The Degenerations of the Kidney are chiefly the fatty and the amyloid. (1.) *The fatty kidney* is large and flaccid, full of yellowish or whitish striæ and marks, combined or not with the characters of the parenchymatous inflammation just described. The calibre of the urinary canals are diminished, and are separated by fatty masses. The glomeruli, and generally the walls of the vascular cells, are fatty or amyloid. (2.) *The amyloid degeneration of the kidney* begins with the arterial capillaries; especially—(1.) The loops of the *Malpighian* tufts; (2.) Their afferent vessels; (3.) The *vasa efferentia* and capillaries in the cortical part; (4.) The "*arteriolæ rectæ;* and after the lesion has advanced to a certain point the injection of the cortical substance becomes impracticable; the arteries become impervious, the cortical substance anæmic, while hyperæmia increases in the pyramids; and hæmorrhage is apt to occur at times, owing to the increased pressure on the inelastic vascular walls, giving rise to reddish-brown streaks or spots. Ultimately the Malpighian glomeruli become converted into solid, homogeneous, translucent globules; and the extension of the disease to the tubes and other tissue produces at length the condition known as waxy kidney. Sometimes the degeneration begins in the capillaries of the *arteriolæ rectæ;* but rarely are the large vessels affected, and it is long before the epithelial tissues degenerate.

The amyloid kidney must be distinguished from the large white and soft kidney described by Dr. Bright.

The amyloid kidney is much harder, more tough, and firmer than the white kidney of Bright. It is not easily torn, which is the case with Bright's white kidney. The surface appears uniform and smooth. The cortical substance is greatly increased in extent, pale, anæmic, and of a dim waxy appearance. The translucency, hardness, and uniformity are sufficient to identify the extreme cases; and in the more common and slighter cases the Malpighian glomeruli can be seen shining prominently, like glistening grains of a whitish-grey appearance, on the cut surface. The iodine test is absolutely necessary; and, unless a microscope has been used, it cannot be declared that amyloid degeneration is not present. Without any re-agent, the Malpighian glomeruli appear under the microscope as globular transparent bodies, with a glistening aspect if they have suffered degeneration.

The capsule of the amyloid kidney is generally easy torn off, and loss of substance or atrophy may be present, as indicated by depressions or hollows of the surface beneath the capsules. The degeneration is most frequently combined with parenchymatous or interstitial nephritis, or the interstitial fatty kidney, especially in syphilitic cases.

With regard to the amyloid kidney, Dr. T. Grainger Stewart, Pathologist to the Royal Infirmary, Edinburgh, has published recently (August, 1864, in *Edin. Med. Journal*) further observations, confirming those referred to in the previous volume (p. 131) of this edition.

In the cases of amyloid degeneration, Dr. Stewart has found great amelioration of the symptoms from the adoption of the following rules:—(1.) To attend to the nutrition of the patient, giving good nutritive food in the form best suited to the individual tastes and powers of digestion; (2.) To give such tonic medicines as may improve the appetite; (3.) To give such hæmatic medicines as control the tendency to anæmia; and among these pre-eminently, the syrup of the *iodide of iron.* The *syrup of the phosphate,* as well as the *syrup of the phosphates of iron, quinia,* and *strychnia* (the formula for which is given at page 62, *ante*), are also, I think, most useful remedies; (4.) In all cases in which a syphilitic infection has been traced, and even in many others, *iodide of potassium* is to be given in moderate and sustained doses. It diminishes the bulk of the liver in a remarkable manner (*Edin. Med. Journal,* August, 1864).

Chief Causes

Section X.—Lesions which Tend to be Localized in the Spleen.

LARDACEOUS SPLEEN—Syn., AMYLOID DISEASE; WAXY SPLEEN.

Latin Eq., *Lien lardaceus;* French Eq., *Dégénérescence lardacée;* German Eq., *Speckige Entartung der Milz;* Italian Eq., *Milza lardacea.*

The chief part of the substance of this organ seems to be made up of distinct translucent Malpighian sacculi closely crowded together (Bennett).

The amyloid degeneration of the spleen is frequently associated with a similar condition of other organs, especially the liver and the kidney. It is a condition characterized by great firmness of the spleen, peculiar waxy-like consistence, and with a distinctness and transparency of the Malpighian sacculi which are not usually very obvious (W. T. Gairdner). Of all the viscera and tissues of the body, the spleen is the one which is comparatively the most frequently affected by the lesion, although it was generally unknown up till 1853-4. Of the bodies examined in the Royal Infirmary of Edinburgh it was observed in 10 *per cent.* of all.

In the most extreme cases of the lesion the spleen is enlarged, and has a swollen aspect. Its weight and density are greatly increased. It feels to the touch like the consistence of wax and lead, and its section shows a dry and smooth surface. After exposure to the air its pulp may become of a bright red colour; and then the Malpighian sacculi appear large and distinct, forming round, colourless, *transparent* granules about the size of a pin's head, slightly prominent, and so hard that they may be picked out by a knife. The pulp is greatly diminished in quantity, and seems in some to be entirely absent; so that the sacculi are crowded together, and the degenerate tissue in their vicinity becomes a continuous mass of a globular form. A small arterial twig can sometimes be seen passing into or through these Malpighian sacculi at their central part.

Microscopic sections are easily made, and their characters are even more striking than those presented to the naked eye. Under a low power (40 to 60 diameters) the Malpighian sacculi appear as large clear spaces of a circular or oval form, surrounded by the dark-red pulp. Under the higher powers (above 250)

the nature of the alteration is seen to consist mainly of an alteration in the normal corpuscles of these sacculi, which are converted into and replaced by masses of a colourless, dense, highly translucent, and homogeneous material, and a careful examination discloses the outlines of irregular cell-forms. But these masses, owing to their roughness, are not easily broken up into the particles which compose them (SANDERS). The translucent parts are very little acted on by re-agents (acids, alkalies, alcohol). Its characters are marked and permanent, so that there is no difficulty at any time in recognizing or identifying the character of the degeneration.

Several varieties of the waxy spleen may be noticed. Two forms at least are distinctly indicated by Virchow, Sanders, and Wilks (although the latter observer says three):—

1. *The Form in which the Malpighian Sacculi are the Structures prominently affected.*—They are sometimes large, and sometimes very small. In the former state we recognize the sago-like granules described by Virchow. In the latter case they are best seen after exposure to the air for a little time, or after having been well washed in water, and then put in alcohol, or into a solution of chloride of zinc, which brings out the waxy appearance very distinctly. In the most extreme cases they never occupy more than about half the bulk of the organ, the intervening pulp-substance being healthy.

2. *A Form in which the Pulp-Substance and Trabeculæ are mainly affected, leaving the Sacculi intact.*—It presents the same waxy consistence, the smooth dry section, and other characters of the lesion; but the sacculi are obscured by the peculiar translucent substance which pervades the pulp, and which looks as if melted tallow had been poured into the trabecular spaces (WILKS).

Histologically, there is no real difference between these two forms, because the corpuscles of the sacculi and of the surrounding pulp-substance are now known to be identical (BUSK and HUXLEY in Wedl, p. 247).

The degeneration seems to commence—(1.) In the arterial capillaries, where the little lateral bulgings are in connection with the Malpighian corpuscles. (2.) These sacculi contain at first normal splenic cells; but at a later stage the contents of the sacculi become irregular and granular, and lymph-corpuscles of a gelatinous appearance become changed into the minute masses of

the waxy substance. (3.) Subsequently the trabeculæ and pulp-struma become affected (BUSK and HUXLEY, as above).

Some cases of albuminoid disease related by Dr. Jenner are evidently of this second nature.

These lesions of the spleen seem to have arrested the attention of Drs. Bright and Hodgkin many years ago. They also recognized the frequent association of diseased lymphatic glands with it. Dr. Bright, indeed, refers to the circumstance as having been originally pointed out by Dr. Hodgkin in vol. xvii. of the *Medico-Chirurgical Transactions.*

SPLENITIS.

LATIN EQ., *Splenitis;* FRENCH EQ., *Splénite;* GERMAN EQ., *Milzentzündung;* ITALIAN EQ., *Splenitide.*

Definition.—*Inflammation of the substance of the spleen.*

Pathology.—A disease which is extremely rare in this country. It is usually limited to certain districts, as Cambridgeshire, Essex, or other paludal places. It is common in the East Indies, especially in the low marshy districts of Bengal, and occurs in the paludal districts of other parts of the world. Now and then it is said to originate from a blow or other accidental violence. It is sometimes seen in children under ten years of age.

The few cases of disease of the spleen occurring in this country will account for its pathology having been little studied. In diffuse inflammation, however, of this viscus, we find it enlarged, of a deep venous colour, and its tissue so softened as to be readily broken down, or even reduced to little more than the consistency of coagulated blood. Diffuse inflammation may terminate by resolution, or pus may form; and in this case one or more abscesses, often containing several ounces of pus, have been formed. The abscesses sometimes make their way to the surface. Dr. Baillie mentions that the spleen has been found in a state of gangrene.

Symptoms.—Acute inflammation of the spleen is seldom seen unless accompanied by ague; and the additional symptoms are probably tumefaction and some pain of the left side, followed perhaps by dropsy.

In chronic affections even abscesses will sometimes form without any marked local symptoms. Dr. Abercrombie gives the case of a gentleman who was dyspeptic, but took a great deal of

nourishment, who was much reduced in strength and flesh, but whose pulse was seldom more than 96 to 100; whose nights were good, though he was occasionally slightly feverish, and who was able, till within a few days of his death, to drive out in his carriage. At length he died, after suffering for two or three days from diarrhœa, but without any suspicion of the spleen being affected. On examination, however, the spleen was found enlarged, and in its centre an abscess containing several ounces of pus.

The more common form of diseased spleen is hypertrophy; and in these cases it can almost always be detected by palpation or percussion, sometimes extending low down into the pelvic region, well over on the right side of the *linea alba*, and extending backwards almost to the spine. In these cases the patient complains of weight and uneasiness, rather than of soreness; his pulse is natural, but the countenance extremely sallow, his person greatly emaciated, his bowels irritable; and these symptoms are for the most part accompanied by œdema of the lower extremities, or by ascites. The most remarkable part of the history of these cases, however, is, that notwithstanding the sallow and emaciated state of the patient, he is often seized towards the close of the disease with hæmorrhage from the stomach and bowels, often so profuse that many pints have been passed or thrown up, greatly exhausting the patient, and rapidly hastening his dissolution.

The course of chronic splenitis is generally long, the patient usually surviving one or more years in the worst cases.

Diagnosis.—Enlarged spleen can only be confounded with encephaloid or other tumor of the abdomen.

Treatment.—Bleeding in *splenitis* has not been found to effect a cure, while mercury has been found for the most part, not only not to be useful, but even to be most pernicious. "I feel," says Mr. Twining, "more anxious fairly to show the baneful effects of mercury in the disease now under consideration, because the instructions usually laid down in the best systems of medicine do not inculcate the avoidance of mercury in any case of enlarged spleen, nor do they advert to the pernicious effects of that state of disease which I have termed vascular engorgement." This gentleman, in further proof of his position, mentions thirteen cases in which the patient either died of mortification of the cheek, the nose, the upper lip, or after having lost all his teeth or

a large portion of the jaw, in consequence of the use of mercury, and, if he survived the employment of this medicine, the spleen remained permanently enlarged. Dr. Voigt, physician to the Danish establishment at Serampore, writes that, although most authors recommend mercury, it is an indisputable fact that a very small quantity, even a few grains, generally occasion a profuse salivation, and so violent an affection of the mouth that mortification sets in, the teeth drop out, the bones become carious, and death ensues. Consequently, mercury and bleeding ought to be little used; and in India, in their stead, a *spleen mixture*, not very dissimilar to that recommended by Celsus, is most in vogue. The best, according to Mr. Twining, consists of the following:—

℞. Pulv. Jalap; Pulv. Rhei; Pulv. Calumba; Pulv. Zingib; Potass. Supertart., ā ā ʒj.; Ferri Sulphat., ℈ss.; Tinct. Sennæ, ʒss.; Aquæ Menthæ Pip., ʒixss. Of this mixture an ounce or an ounce and a half is to be taken twice a day, or such quantity as may produce three or four stools in the twenty-four hours.

The spleen mixture is, in some instances, efficacious, but in a much larger number of instances it entirely fails; and under these circumstances the *iodide of potassium* and the *bromide of potash* have been recommended. The dose of the bromide of potash is gr. v. to x. *ter die*, combined with *camphor mixture*. The marked influence of the *biniodide of mercury*, in the form of an ointment, rubbed into the skin over the surface of the spleen, in reducing its enlargement, has been already noticed (p. 828, *ante*). The invalid soldiers who suffer from enlarged spleens invariably apply for some to take away with them when they are discharged from Netley Hospital.

HYPERTROPHY OF THE SPLEEN.

Latin Eq., *Hypertrophia;* French Eq., *Hypertrophie;* German Eq., *Hypertrophie;* Italian Eq., *Ipertrofia.*

The spleen is more often hypertrophied, as a result of malaria or of *leucocythæmia*. In the *Medical Commentaries* a hypertrophied spleen is mentioned which weighed 11 lbs. Portal speaks of another that weighed 30 lbs.; and Lieutaud met with one, in a woman who had been ill seventeen years, that weighed 32 lbs. It is singular that these large tumefied spleens sometimes subside

very rapidly. Abercrombie mentions one that subsided in a week after the ague on which it depended had been arrested. The hypertrophied spleen is generally more or less indurated.

The spleen is occasionally atrophied, so that little more than a rudimentary spleen remains. It is also found indurated, and often greatly softened. Hydatids have been found in the spleen. In a few instances small portions of the spleen, about the size of a nut, are found indurated and nearly white. These appearances are supposed to arise from slight effusions of blood into the substance of the spleen, which become organized, and the colouring particles being absorbed, leave the appearances in question; or they are due to *emboli*.

CHAPTER XIV.

DISEASES OF THE CUTANEOUS SYSTEM.

Section I.—General Pathology and Classification of Diseases of the Skin.

Diseases of the skin have been regarded too much as a specialty; and only now are they beginning to be looked upon as a class of diseases whose pathology is capable of being investigated and studied like other diseases. The expressions of skin diseases are undoubtedly various in appearance; and the same disease does not always exist in the same simple or elementary form.

The classification of skin diseases hitherto in use is that which is comprehended in the eight orders of Willan and Bateman; and the characteristics of these orders are embraced in the following definitions of terms in common use in the description of skin diseases:—

Order I. Pimples.—*Papulæ* are simple solid acuminated elevations of the cuticle, resembling an enlarged *papilla* of the skin. They commonly terminate in a scurf, and sometimes, though seldom, in slight ulceration of its summit.

Order II. Scales.—*Squamæ* consist of cuticle in patches, plates, or laminæ, in which the epidermic cells are morbidly adherent, hard, thickened, whitish, and opaque. These scales cover either small papillæ, red elevations, or larger deep red and dry surfaces.

Order III. Rushes.—*Exanthemata* are composed of superficial red patches

of irregular size, and variously diffused. They disappear under pressure, and terminate by desquamation.

ORDER IV. BLEBS.—*Miniature Blisters—Bullæ.* They differ from vesicles in size, being larger. A large portion of cuticle is detached from the skin by the interposition of a watery fluid, usually transparent. The skin is red and inflamed beneath the blebs.

ORDER V. PUSTULES.—*Pustulæ* consist in circumscribed elevations of the cuticle, and contain pus. They have red and inflamed bases, and are succeeded by an elevated scab, which may or may not be followed by a cicatrix.

ORDER VI. VESICLES.—*Vesiculæ*—small acuminated or orbicular elevations of the cuticle, containing lymph, which, at first clear and colourless, may become amber-coloured, opaque, or pearl-like. Vesicles are succeeded by a scurf or a laminated scab.

ORDER VII. TUBERCLES.—*Tuberculæ*—small, hard, indolent elevations of the skin, sometimes suppurating partially, sometimes ulcerating at their summit.

ORDER VIII. SPOTS.—*Maculæ* are permanent discolorations or stains of some portions of the skin, often with a change of structure. They may be whitish, dusky, or dark.

Under these several orders Dr. Bennett proposes the following classification:—

ORDER I.—*Exanthemata.*
- Erythema.
- Roseola.
- Urticaria.

ORDER II.—*Vesiculæ.*
- Eczema.
- Herpes.
- Scabies.
- Pemphigus.

ORDER III.—*Pustulæ.*
- Impetigo.
- Ecthyma.
- Acne.
- Rupia.

ORDER IV.—*Papulæ.*
- Lichen.
- Prurigo.

ORDER V.—*Squamæ.*
- Psoriasis.
- Pityriasis.
- Ichthyosis.

ORDER VI.—*Tuberculæ.*
- Lepra Tuberculosa.
- Lupus.
- Molluscum.

ORDER VII.—*Maculæ.*
- Lentigo.
- Ephelides.
- Nævi.
- Purpura.

ORDER VIII.—*Dermatozoa.*
- Entozoön folliculorum.
- Acarus.
- Pediculus.

ORDER IX.—*Dermatophytæ.*
- Achorion Schönlein (Favus).
- Achorion Grubii (Mentagra).

These and similar classifications are mainly anatomical. They do not attempt to throw any light on the causes producing the various diseases, which are contemplated as so many distinct and individual "unities," their mutual relations being of secondary importance. The basis of classes, as proposed by Willan and Bateman, is only useful as furnishing outward marks or anatomical characters useful in describing the morbid anatomy of skin diseases; but it has no relation to the causes, to the pathology, nor to the treatment of such diseases. An affection which is papular to-day may be vesicular to-morrow, and pustular eventually.

Under the same division, or class, maladies are brought together which Nature has stamped with broad and obvious marks of distinction. Febrile diseases are associated with non-febrile; and ailments which are local and trivial are associated with diseases of grave import and deeply rooted in the system. And, on the other hand, distempers which Nature has plainly brought together and connected by striking analogies and resemblances, the methodical arrangement of Willan and Bateman puts widely asunder (WATSON).

A purely anatomical classification of cutaneous lesions may be stated as follows, after the late Dr. A. B. Buchanan:—

I. **Deformities of the Skin** (mostly persistent and non-inflammatory).

II. **Eruptions of the Skin** (mostly transient, and with the phenomena of inflammation).

(A.) PRIMARY FORMS.—(1.) *Macula:* (*a.*) Erythematous; (*b.*) Purpurous—comprehending stigma, petechia, vibix, ecchymosis. (2.) *Papula.* (3.) *Tuberculum.* (4.) *Pomphus.* (5.) *Vesicula*—comprehending vesicula miliaris and phlyctæna. (6.) *Pustula*—comprehending achor, psydracium, and phlyzacium. (7.) *Bulla.*

(B.) SECONDARY FORMS.—(1.) *Excoriatio.* (2.) *Ulcers.* (3.) *Rima.* (4.) *Squama.* (5.) *Crusta.* (6.) *Cicatrix.*

Alibert conceived the idea of arranging skin diseases into natural families, of which he gives *twelve.* His classification presupposes a knowledge of the subject incompatible with teaching.

M. Hardy, of the Hôpital St. Louis, classifies skin diseases, according to their nature, into the following *ten* natural families:—(1.) *Macules and deformities;* (2.) *Local inflammations;* (3.) *Parasitic diseases;* (4.) *Eruptive fevers;* (5.) *Symptomatic eruptions;* (6.) *Dartres, or Tetters;* (7.) *Scrofulides, or strumous eruptions;* (8.) *Syphilides, or syphilitic eruptions;* (9.) *Cancers;* (10.) *Exotic diseases.*

In an admirable paper "On the Theory and Classification of Inflammations of the Skin," by the late Dr. A. B. Buchanan, Physician to the Dispensary for Skin Diseases in Glasgow, it is shown that, in the present state of our knowledge, any classification resting on one principle of division only, runs the risk of being, to a greater or less extent, *artificial* and untrue; and to secure a *natural* system, several principles must be taken into account, though greater importance may be attached to some of these than to others (*Edin. Med. Journal,* January, 1863). Skin diseases, like all other diseases, ought to be classed according to their

nature or *pathology*. The cause of a disease, when known, gives a more accurate indication of its true nature, and its means of cure, than a knowledge of its anatomical appearances or symptoms, which are mere effects of the cause. So far as can be done, therefore, Dr. Buchanan proposes that skin diseases should be classified according to their causes. Not knowing the cause, some other principles must be sought for under which groups may be formed; and he finds—in the pathological processes recognized as *inflammations, new formations, hæmorrhages*—that skin diseases may be arranged into three or more groups.

The inflammatory skin diseases are those which affect the "*pars papillaris*" of the corium, as well as the "*pars reticularis*" and the subjacent connective-tissue; but the process is not confined to these, for it affects even the epidermis, whose cells are changed in formation and in structure in the same way as all cells are altered by the process of inflammation.

The skin diseases characterized by new formations may be regarded as—(1.) The productive effects of inflammation; or (2.) The productive effects of such constitutional states as cancer, scrofula, and Bright's disease.

The hæmorrhagic skin diseases arise from the escape of blood in small quantities, owing to the rupture of capillary vessels, or to the escape of colouring matter along with the exudations. They are affections peculiar to the cutis; for, although blood may be effused among the epidermic cells, hæmorrhage can only originate from the cutis.

On these principles a classification somewhat of the following form has been suggested:—

Classification of Skin Diseases (Dr. A. B. Buchanan).

Class I.—Inflammations.

Group I.—Simple Inflammations (allied to *Simple Dermatitis*).

(1.) Erythema ([a.] *simplex;* [*b.*] *multiforme;* [c.] *chronicum*—comprehending *papulatum, nodosum, strophulus, squamosum, pityriasis furfuracea, membranacea, rubra*).

(2.) Herpes (*simplex* and *zoster*).

(3.) Urticaria (idiopathic, from ingestion of particular kind of food; from uterine affections; or persistent).

(4.) Dermatitis (idiopathic, as from *burns*, or from *frost-bite;* or symptomatic, as of *erysipelas;* or phlegmonodes, as *furunculus, anthrax, Aleppo tubercle*).

(5.) Pemphigus (benign, persistent, and foliaceous).

GROUP II.—ECZEMATOUS INFLAMMATIONS (ALLIED TO *Eczema*).

(1.) ECZEMA (*erythematodes; E. papulosum*, comprising *lichen simplex* and *prurigo; E. vesiculare; E. rubrum; E. pustulosum*, comprising *impetigo sparsa, figurata*, and *pilaris; E. lichen; E. squamosum; E. pityriasis*).
(2.) ACNE (comprising *A. simplex; A. pilaris; A. rosacea*).
(3.) ECTHYMA ([a.] *simplex;* [b.] *chronicum = rupia;* [c.] *gangrenosum*).
(4.) PSORIASIS (*punctata, guttata, nummularis, circinata* [lepra], *gyrata, confluens*).

GROUP III.—ULCERS.

(1.) IDIOPATHIC.
(2.) SYMPATHETIC.
(3.) CONSTITUTIONAL.

CLASS II.—New Formations.

GROUP I.—HOMOLOGOUS NEW FORMATIONS.

(1.) EPIDERMIC (*epithelial growths*, comprising *callositus, clavus ichthyosis, cornu cutaneum*).
(2.) PIGMENTARY (*lentigo, ephelis, moles, melanosis, chloasma, silver stain, leucopathia*).
(3.) DERMIC (*cicatrix normal*, or *cheloid, cutaneous tumor* [wens], *multiple tumors* [mycosis], *molluscum simplex, condylomata, verruca vulgaris, verruca mollis*).

GROUP II.—HETEROLOGOUS NEW FORMATIONS.

(1.) PSEUDOPLASMS (*lupus*, comprehending *maculosus, tuberculosus, hypertrophicus, exedens, serpiginosus;* and *lepra*, comprehending *maculosa, tuberculosa anæsthetica, exulcerans*).
(2.) NEOPLASMS (*epithelioma, carcinoma*).

CLASS III.—Hæmorrhages—*e. g., petechiæ, vibices, ecchymosis, purpura.*
CLASS IV.—Diseases of Accessory Organs—e. g., *hair, nails, sweat glands.*
CLASS V.—Diseases Defined by Uniform Causes.

GROUP I.—PARASITIC DISEASES.
GROUP II.—SYPHILITIC ERUPTIONS.
GROUP III.—ERUPTIONS OF SPECIFIC FEVERS.
GROUP IV.—SCROFULODERMATA.

SECTION II.—DESCRIPTION IN DETAIL OF THE MORE COMMON DISEASES OF THE SKIN.

ERYTHEMATOUS INFLAMMATIONS.

1. *Erythema.*—Its expression is that of uniform redness simply, with puffiness of the skin, distributed in distinct patches of some size. It is accompanied by little constitutional disturbance; and if febrile phenomena are decided, it may betoken more severe areolar inflammation than *erythema*, and a more grave disease. It is thus apt to be mistaken for *erysipelas.* The varieties of Erythema are,—*E. lœve; E. fugax*—syn., *E. volaticum; E. mar-*

ginatum; E. papulatum; E. tuberculatum; E. nodosum. Of these varieties *erythema nodosum* is perhaps the most important. The indisposition which precedes the eruption is generally associated with a slight degree of fever. Red oval patches, considerably elevated and very tender, appear on the fore part of the legs, sometimes on the arms; and the long diameter of these patches is generally parallel to the axis of the limb. They sometimes form bumps an inch and a half long and an inch broad on the anterior aspect of the leg. After a few days the red colour changes to a blue, the patches become soft, and although something like fluctuation may be felt, yet suppuration does not occur. Thus the bumps of erythema gradually subside. Sometimes the disease is seen in feeble boys; but it is most common in young women, in whom it seems associated with disordered menstruation, or with rheumatism (RAYER, WATSON). In the chronic stage desquamation invariably occurs; and to this stage of the disease the name *pityriasis* has been given.

Treatment.—*Rest* and *quinine*, after aperients.

2. *Herpes.*—This disease is expressed by red patches, of irregular form and variable size, upon each of which there arises a group, cluster, or crop of extremely minute vesicles. It is often preceded by considerable local irritation, or by cutaneous pain or neuralgia, which sometimes remains after the eruption has died away. The eruption runs a very definite course, and, when not interfered with, its development and defervescence is completed in about ten days. The vesicles ultimately rupture, and a gum-like scab forms over the group, which shrivels and contracts upon itself. The disease occurs sometimes—(1.) As one or two patches on the lips—*herpes labialis;* (2.) As a band of patches which encircles half the circumference of the trunk, or the whole circumference of a thigh —*herpes zoster, zona ignea, cingulum,* or *the shingles;* (3.) A very common and troublesome form of the disease affects the foreskin—*herpes preputialis.* Its effects are often mistaken for chancres, and by unprincipled persons may be treated as such. It has nothing to do with syphilis, and its history is a sufficient guide to diagnosis (see vol. i., p. 759). The disease may be prolonged by ulceration of the vesicles, or by irritant applications, or when the scabs are prematurely chafed off.

Treatment.—The state of the stomach and digestive organs, and diet generally, should be regulated. Malt liquors should be

avoided; and, if pain is severe, opiate fomentations may be applied. *Herpes preputialis* requires no treatment beyond careful ablution with tepid water, and the interposition of a piece of wet lint between the prepuce and the *glans penis*. If there is much pain, the lint should be moistened with a watery solution of opium.

3. *Nettlerash*, or *Urticaria*, consists of an eruption of little solid elastic eminences, roundish or oblong, pale in the centre, and red at the circumference. These are commonly called "wheals," similar to what results from the mark of a lash. The eruption is attended with an intense heat, a burning, tingling, or pricking sensation, very much like that produced by the stinging of a nettle. There are several varieties of *urticaria*—(a.) *Urticaria acuta;* (b.) *Urticaria chronica*. The acute forms are generally connected with the ingestion of some kinds of food—oatmeal, bitter almonds, shell-fish, &c. The more chronic and intermittent forms are associated with uterine or other affections.

Treatment.—Emetics and purgatives in the first instance; afterwards the correction of faulty digestion. The surface of the eruption may be dusted over with flour; or the following lotion may be used:—

℞. Carbonatis Ammoniæ, ʒi.; Plumb. Acetatis, ʒii.; Aquæ Rosearum, ℥viii.

4. *Pemphigus*, or *Pompholyx*, is characterized by an eruption of large vesicles, filled with serous fluid, known as *bullæ*. These *bullæ* vary in magnitude, are generally distinct, but numerous. They spring up in successive crops, generally in the forearms and legs, surrounded by redness of the skin. The vesicles, originally transparent, gradually become opaque, pearl-coloured, and ultimately of a pale red colour. Usually the disease is prolonged over weeks, months, or years, and is not attended by any decided febrile disturbances. A slight pricking sensation indicates a red spot, where the eruption commences. In the centre of each of these spots the cuticle begins to rise like a blister; and as the circumference of the redness increases, the blister-like vesiculæ are so rapidly formed that, in a few hours, each vesicle may be as big as a hazel-nut or walnut. The *blebs* are then apt to burst, when a straw-coloured serum exudes; and the epidermis contracts into folds or wrinkles, the surface underneath being red, painful, and smarting. If the vesicles do not burst, small brownish, flat crusts form.

The disease is usually associated with debility, intemperance, and bad or insufficient food. The following varieties are to be distinguished:—(a.) *Pemphigus acutus;* (b.) *Pemphigus chronicus;* (c.) *Pemphigus solitarius;* and (d.) *Pemphigus foliaceous.*

Pemphigus foliaceous commences on the front of the chest, and when fully developed covers the whole body. It is almost always fatal.

Treatment.—Dietetic and tonic, especially quinine and the mineral acids. Arsenic is said to be of use in obstinate cases. Emollient lotions, with or without opium, are useful local applications.

ECZEMATOUS INFLAMMATIONS.

Eczema, in some of its many forms, is one of the most common of skin diseases. The eruption, when at its height, is characterized by—(1.) Infiltration of the skin; (2.) Exudation on its surface; (3.) The formation of crusts; (4.) Itching. Eczematous skin feels thick when pinched up into a fold. It has a doughy feel, and on pressure the redness disappears, and shows a yellowish hue of the skin. The exudation is watery, sometimes purulent, and sometimes mixed with blood. It stains and stiffens underclothing with which it may come in contact. The exudation becoming concrete, forms crusts, and their appearances vary with the character and nature of the exudation. The itching is always aggravated by touching the part, and still, at the same time, an irresistible desire to scratch is excited. Scratching is sometimes thus persevered in till blood flows from the part. The disease is thereby greatly aggravated—a copious eruption of newly formed vesicles and fissures being apt to form.

The elementary lesions of the skin in eczema are—(1.) An erythematous state of the skin; (2.) Vesicles; (3.) Pustules; (4.) Papules; (5.) Fissures; (6.) A mixture of several or of all of those lesions; and in the fully expressed disease there may not be the merest vestige of either a vesicle, a pustule, or a papule, but the skin is red and smooth on the surface, having a brilliantly polished and shining appearance, the tissue being loaded with infiltration. The vesicles, when they occur, are usually developed at the orifices of the cutaneous follicles. They are small and closely set together: they usually rupture early, and the serosity concretes into crusts. The more the skin is

infiltrated the less likely are vesicles to be seen. In the absence of vesicles, when the disease is at its height, the infiltrated patches are red and inflamed. The redness is not uniform: it is studded with innumerable points of a deeper red, giving a punctated appearance to the part (DEVERGIE). These points correspond to the orifices of the glands, like the vesicles which may have preceded them; and the appearance is due to the greater congestion of the skin which surrounds the granular orifices. Minute excoriations are apt to occur at these spots, the result of rupture of the vesicles. From these excoriated points serous fluid continues to exude, which afterwards concretes into scabs (*eczema vesiculorum*). The *impetigo* of authors is the pustular form of *eczema*, and the pustules form upon erythematous patches of skin, just as the vesicles did before them. They are most common on the head and chin. Vesicles and pustules are often seen on the same patch (*eczema impetiginodes* or *eczema pustulosum*).

Lichen is the papular form of *eczema*, the eruption commencing as small red papulæ, either isolated or confluent. These becoming excoriated, give vent to a serous fluid in considerable abundance, which ultimately concretes into a crust. If the summits of the papulæ become torn by the nails in scratching, yellow or blackish crusts form, due to the mixture of blood and serum, and abrasions of the skin occur. The disease is then known as *prurigo* (*eczema lichenoides* or *eczema pruriginosum*). It occurs more or less in all chronic cases of *scabies* or *phthiriasis*, and sometimes in *urticaria*. The disease attacks by preference the scrofulous and the debilitated, and is especially associated with improper, insufficient, or bad food. A too liberal diet, and stimulants in food or drink, similarly predispose to the disease. Exposure of the skin to the heat of the sun, or to acrid substances, also brings about the disease—*e. g.*, in cooks, grocers, bakers, and the like. Fissures of the skin are a most frequent complication, and occur in those situations where the skin is naturally thrown into folds, as at the arms, the angles of the mouth, the joints, the palms of the hands, and flexures of the fingers. They are apt to increase in depth as the disease continues, sometimes bleeding, and causing excruciating pain. When deep, the fissures are red and raw-looking, and serum or blood exudes from them, giving rise to crusts which partially fill them up (*eczema rimosum* of T. M. Anderson and Buchanan—the *eczema fendillé* of the French).

Any part of the skin may be the seat of *eczema;* but there are certain localities where it is most apt to occur. These are the head, the hairy portions of the face, the lips, the edges of the eyelids, the nostrils, the lips, the external auditory passages and ears, the hands, feet, legs, genitals, perineum, *mammæ*, umbilicus, and parts of the skin which are naturally in contact with one another.

On the head and hairy parts of the face the disease occurs most frequently in the pustular form (*impetigo capitis*); and collections of pus are apt to form beneath the crusts and scabs, attended sometimes by little cutaneous abscesses and enlargements of the lymphatic glands. Each pustule of eczema is situated at the orifice of a hair follicle; and the hair may be seen to pass through the centre of each pustule. These pustules dry up into small yellow crusts when the disease affects the hairy parts of the face; and the skin on which the pustules are developed assumes a dusky red tint, becoming gradually more thickened and infiltrated (*impetigo menti—eczema pilare faciei*). In the *eczema* of the internal auditory passages the disease usually affects the auricles, commencing on the skin of these parts, and gradually extending inwards. The calibre of the meatus is narrowed, and often so much so that it is impossible to see the *membrani tympani.* A milky or watery fluid exudes from the meatus, and moistens the pillow at night. The discharge is apt to have a very bad odour if the ear is not frequently washed out (Anderson, *Med. Times and Gazette,* June, 1863).

Treatment of eczema must be constitutional in the first instance; and local applications suited to the nature of the part affected are to be carefully used. Derangements of the digestive organs must be especially rectified. If the tongue is loaded, the appetite bad, the liver torpid, as indicated by light, clay-coloured evacuations and costive bowels, small doses of grey powder in combination with quinine are indicated. Dr. T. M. Anderson suggests the following:—

℞. Sulphatis Quiniæ, gr. xii.; Pulv., Rhei., gr. xxxvi.; Hydrarg. c. Cretâ, gr. xxx.; Sacchari Albi, ʒi.; *misce, et divide in Pulv.* xii.

Two of these powders are to be taken daily by an adult; but at any age the dose must be so adjusted that the patient has at least one full natural evacuation daily. If the patient is robust, but

with the functions of the liver and bowels impaired, occasional doses of *calomel*, alone or in combination with *scammony*, will stimulate the torpidity of the digestive organs; and at the same time the cutaneous inflammation will diminish. If the patient is a full feeder, and will not be persuaded to live more sparingly, *one drachm to two drachms of the sulphate of magnesia* may be given twice daily, with *a sixth to half a grain of tartar emetic* added to each dose. The effect of this remedy will be to diminish desire for food, and at the same time keep the bowels freely open. If the patient is scrofulous, or debilitated from insufficient food, or food not nutritive enough, more nourishing food must be given, combined with tonics containing iron and cod-liver oil. Children a few months old, reduced to "skin and bone," recover wonderfully under the influence of twenty drops of the syrup of the iodide of iron in a tea-spoonful of cod-liver oil repeated three times daily, the dose of the oil being gradually increased to a table-spoonful (ANDERSON, l. c., June 27, 1863). *Syrup of the phosphate of iron*, or of the *phosphates of iron, quinia*, and *strychnia*, should be alternated with the *iodide of iron*.

The treatment must be steadily maintained for at least six weeks or two months. In severe cases the oil may be rubbed into the skin, in addition to giving it internally. Cod-liver oil and iron are almost equally serviceable to eczematous adults. When there is a difficulty of taking it in its pure state, the emulsion of it will often be found useful;—when the appetite is very deficient, a pure tonic, such as *quinine* and *aromatic sulphuric acid*, as in the following:—

℞. Sulphatis Quiniæ, gr. xvi.; Acid. Sulphurici Aromatici, ℥iv.; Syrupi Limonum, ℥ss.; Infus. Cascarillæ, ad ℥viii.; *misce, et cola per chartam.* A table-spoonful to be taken three times a day half an hour before food.

If the eczematous patients are robust and plethoric, the local abstraction of blood by leeches or scarification is often beneficial; but in general the action of *calomel purgation* is sufficient to reduce the inflammatory action, combined with a regulated abstemious diet of mixed animal and vegetable food simply dressed. Dishes of pastry, pickles, spices, strong tea, and coffee, are particularly to be avoided; while the use of wine, spirits, and malted liquors must be entirely suspended.

If the patient has been much in the habit of using stimulants, the eczema will be much more difficult to subdue than if he had been an abstainer from these fluids. It is sometimes necessary to prescribe milk diet for a time, all animal food being proscribed.

Of internal medicines, arsenic, sulphur, and the alkalies are especially useful (STARTIN, ANDERSON). Fowler's solution (the *liquor arsenicalis* of the Edinburgh Pharmacopœia) is well suited. An adult may commence with *five* minims thrice daily; and at the end of a week the dose should be increased by *one drop* every second or third day, till the disease begins to yield, or the medicine to disagree (ANDERSON). "In order to secure the virtues of arsenic as an alterative, it will be necessary to push the medicine to the full development of the phenomena which first indicate its peculiar action on the system. Arsenic as a remedy is too often suspended, or altogether abandoned, at the very moment its curative powers are coming into play. The earliest manifestation of its physiological action is apt to be looked upon as its poisonous operation; and the patient declares that the medicine has disagreed with him. Forthwith the physician shares his fears; the prescription is changed, and another case is added to the many in which arsenic is said to have failed after a fair trial of its efficacy" (Begbie, *Contributions to Practical Medicine*, p. 270). *Arsenical solutions* should be given immediately after food; and in persons whose digestive organs are weak, a tonic infusion, such as the infusion of *cascarilla* or *gentian*, is the best vehicle for its administration; and a few drops of morphia may sometimes be added, as in the following:—

℞. Solut. Fowleri; Solut. Muriatis Morphiæ, ā ā ʒii.; Syrupi Limonum, ʒss.; Tinct. Cocci Cacti, ʒss; Infus. Cascarillæ, ad ʒxii.; *misce.* A table-spoonful of this mixture to be taken thrice daily immediately after food.

The *liquor sodæ arsenias*, in doses of five to ten minims, is said to cure eczema with less gastric disturbance and with less irritability of the conjunctiva than the *liquor arsenicalis*. As the disease yields, the dose may be diminished; but the use of the remedy should not be suspended till some time after the complete removal of the eruption. In the case of infants at the breast, arsenical solutions may be given to the mother; and *mercury may be combined with the arsenic, as in Donovan's solution, of*

which ten minims may be given thrice daily. The use of the natural mineral waters which contain sulphur is the best method of securing the full effects of the alterative action of sulphur on eczematous patients. These waters are especially Harrowgate and Moffat in this country. *Alkalies* are most beneficial in those cases where the patient has been in the habit of taking stimulants; and when there is a tendency to acidity of the stomach, and to the deposit of *lithates* in the urine, or to gout, or to rheumatism, *aqua potassæ may be given, largely diluted with water, in doses of twenty minims three times a day.* Dr. Anderson recommends *sesquicarbonate of ammonia* (now called the *carbonate of ammonia* in the *British Pharmacopœia*), in doses gradually increasing from ten to thirty grains. If gout prevails, *colchicum* ought to be given with the other remedies; and in rheumatism the *acetate* or *bicarbonate of potash* in half-drachm doses should be added to each dose. All the alkaline remedies should be largely diluted with water.

With regard to local treatment, the first point is to remove all sources of irritation, and especially the crusts. A poultice composed of *crumbs of bread* and *hot almond oil* applied to the eruption at night will usually bring away the crusts in the morning. If they fail to be detached by this application, they must be again lubricated with fresh almond oil, and forcibly removed when they are thoroughly softened. If, after the crusts are removed, the surface is seen to be acutely inflamed, and if there is a *burning heat* in place of *itching*, local sedatives must be applied. For this purpose Dr. Anderson recommends a cold potato-starch poultice, a small quantity of powder containing camphor being sprinkled over the inflamed surface before the poultice is applied. The camphor powder may be composed of the following ingredients:—

℞. Camphoræ, ʒss.; Alcoholis, q. s.; Pulv. Oxydi Zinci; Pulv. Amyli, ā ā ʒiii. This powder must be kept in a stoppered bottle (Anderson).

Instead of poultices, a mixture of powdered oxide of zinc and glycerine, in the proportion of half an ounce of the one to two ounces of the other, will be found to be a soothing application; and to it a little camphor may also be added, as in the following:—

℞. Camphoræ, ℈ii.; Pulv. Oxydi Zinci, ℥ss.; Glycerinæ, ℥ii.; Cochinillini, gr. ii.; Spt. Rosmar., ℥i.; *misce.* This mixture must be stirred before using it, a thin layer being then rubbed over the inflamed part twice or thrice daily (ANDERSON).

When the disease passes into a chronic stage, as indicated by the disappearance of the burning heat and the supervention of itching, the local applications must vary with the condition of the parts. If there be much *infiltration* of the tissues, treatment by potash applications is the most efficient (HEBRA, ANDERSON). The more extensive the disease the *weaker* the solution ought to be for application over the large surface. If the infiltration is slight, common *potash soap* (soft soap, black soap—*sapo mollis*, *sapo viridis*), or a solution of *one part of it in two of boiling water a little oil of rosemary or citronella being used to conceal the odour*, may be used. A piece of flannel dipped in this liniment should be rubbed as firmly as possible over the affected parts night and morning, the solution being allowed to dry upon them. It should, however, be washed off after each application. If the patient can bear it, a piece of flannel, saturated with the solution, may be left in contact with the part all the night. Another method of treatment is to paint over the eruption night and morning with a large brush, charged with the *aqua potassæ* of the *Edinburgh Pharmacopœia*, its irritant action being neutralized by means of cold water when the smarting becomes excessive. A solution of *potassa fusa* may also be employed of various strengths. If the case be mild, *two grains* to an *ounce* of water; if more severe, then *five, ten, twenty, thirty,* or *even more grains to the ounce of water* may be used. *But all these stronger solutions must be washed off very speedily, and they ought not* to be employed more than once daily. Such remedies the physician ought to apply himself, and not entrust the control of their action to the patient. If there is a tendency to the formation of fissures, which are apt to result from the use of potash, *cod-liver oil* or *glycerine* should be applied to the parts every night. As infiltration subsides, the solution ought to be gradually diluted; and great caution is necessary in using such local applications upon infants, delicate females, and old or infirm persons. The affected parts should be bathed repeatedly during the day in cold water, during the use of these *potash* applications. The *cold douche* may be employed where it is practicable. If the itching is very intolerable, *prussic acid*

may be mingled with the potash applications, as in the following:—

℞. Potassæ Fusæ, gr. x.; Acid. Hydrocyan. Dil. (Ed. Ph.), ℈i.; Aq. Rosarum, ℥ii.; *misce.* A little of this solution is to be rubbed firmly over the eruption night and morning, and at any time when the itching sensation is severe.

Tarry applications are of great value in the local treatment of the *declining stages* of eczema, when infiltration and itching are subdued. Common tar (*pix liquida*) or oil of cade (*oleum cadini*, as manufactured at Aix-la-Chapelle) should be rubbed firmly over the eruption by means of a flannel cloth, and allowed to dry upon it. It should be applied thrice daily, and washed off with *soft soap* or *petroleum soap.* Dr. Anderson recommends that common tar should be combined with one of the potash solutions—for example, a mixture of *equal parts of common tar, methylated spirit, and soft soap,* used as above directed, will be found useful, or the following preparation, as less offensive:—

℞. Saponis Mollis, Spt. Rectificati, Olei Cadini, ā ā ℥i.; Olei Lavandulæ, ʒiss.; *misce.* A little quantity is to be firmly rubbed over the eruption night and morning; and it must be washed off before each re-application.

Of *mercurial applications* the *citrine ointment* (*Ung.-Hyd. nit.*) is the best, or *the ointment of the iodide of mercury.* They may be used of their full strength, or diluted with lard, according to the indications of the case, care being taken that *mercurialism* is not induced. In using ointments, a small quantity should be melted on the finger, and rubbed firmly into the affected part; and none should be allowed to lie undissolved upon the skin; and the part should always be cleaned with soap and water before re-application. Epilation should be resorted to in cases of eczema of the hairy parts of the face. In very mild attacks, or with a view to prevent the recurrence of the disease, the skin may be washed occasionally with soft soap and water. Hendrie's "dispensary petroleum soap" is recommended by Dr. Anderson, to whose admirable lectures on "Eczema," in the *Medical Times and Gazette* for May, June, and July, 1863 (since published in a separate volume), the reader is referred for more detailed

information regarding the nature and treatment of this inveterate skin disease.

Achne affects the subaceous follicles of the skin generally, or those at the roots of the hairs of the beard. The disease generally appears as pimples; but suppuration usually very slowly follows: there may be persistent redness after suppuration is at an end, giving a blotchy appearance to the part. The parts most frequently affected are the temples, cheeks, nose, and forehead; but it also appears on the neck, shoulders, and front of the chest. The first occurrence of the disease is often indicated by the collection of morbid material in the subaceous follicles, which open on the skin by a blackish point, to which the vulgar give the name of *"grubs"* or *"worms" in the skin.* The tubercles on which the classification of this disease is sometimes based are merely the terminations of the pustules, and not an elementary lesion. The accumulation of the matter secreted by the subaceous follicles tends to keep up the eczematous inflammation in them.

Treatment.—Local and general measures require to be combined. Diet, in the first instance, should be restricted. Wine, spirits, and coffee are to be refrained from. Milk is to be used as a drink, and as an article of diet, combined with light food, fresh vegetables, and succulent ripe fruits. Emollient applications, such as an emulsion of bitter almonds, a decoction of bran or quince seeds, and tepid milk, are useful. Dr. Anderson has found one or other of the two following lotions particularly serviceable; and generally, if one fails, the other succeeds:—

℞. Sulphuris, ʒi.; Spt. Rectificati, ℥i.; *misce.*

The mixture is to be shaken before using it; or,

℞. Hyd. Corrosiv. Sublim., gr. ix.-xii.; Hydrochl. Ammoniæ, ʒss.; Cochinillini, gr. i.; Aq. Rosæ, ad ℥vi.; *misce.*

Whichever of these lotions is used, let it be applied by dipping a piece of flannel into the lotion, and rubbing *very firmly* over the eruption night and morning.

To promote resolution of the induration, *iodide of sulphur,* in the proportion of *fifteen* or *twenty-four grains to an ounce of lard,* is of great service in dispelling the tumors. Drastic purgation ought to be avoided. Simple baths at a temperature of 88° or 90° Fahr. are of service.

Ecthyma is characterized by large round pustules, generally

distinct, and seated upon a hard inflamed base. The pustules are succeeded by dark-coloured scabs, which leave superficial cicatrices behind them, or red stains, which disappear after a time. The eruption of ecthyma is common on the neck, shoulders, buttocks, extremities, and chest; and is seldom developed on the face or scalp. The typical appearance of ecthyma may be seen in the pustules produced by friction with *tartar emetic* ointment. The pustules continue for several days, and are succeeded by scabs, which begin to form in the centre. The disease may occur at any age or season, but it most frequently appears during spring and summer in young adults. The *ecthyma cachecticum* of Willan prevails in old, irritable persons much addicted to alcoholic fluids.

Treatment is chiefly by diluents, simple and emollient baths, and regulation of diet. Moderate exercise should be taken, combined with the use of alkaline or salt water baths. Mild laxatives are beneficial, and spirits, wine, and beer are to be refrained from. The food should be as nourishing as can be digested. Tonics, such as quinine and iron, are also indicated. A stimulating lotion, composed of *muriatic acid* diluted with water, is of use to brush over the parts and promote cicatrization.

Psoriasis vulgaris.—Psora leprosa, Lepra vulgaris, or *dry tetter,* is characterized by the development of irregularly formed patches, slightly raised above the level of the skin, and covered with thin, dry, white scales. The patches may be distinct, small, and scattered; or larger, confounded together, and irregular; or they may be so extended as to make a continuous surface. Hence the names of varieties of psoriasis—*guttata, diffusa, gyrata, inveterata.* The intense itchiness and the eruption are always preceded and accompanied by that form of dyspepsia, or impaired digestion, in which there is a superabundance of acidity, much formation of lithates, and an obvious constitutional tendency to gout. Anæmic persons, and those in whom the circulation is languid, with a dry skin, are those in whom the disease is prevalent. Sometimes it is localized in patches, as on the back, between the shoulders, the lips, the eyelids, the palms of the hands, the scrotum, or the pudenda. Sometimes it seems hereditary. Intemperance, the use of highly salted food, fish, or indigestible substances, are apt to induce an attack. The depressing passions, anxiety and grief, are also often followed by *psoriasis.*

Treatment.—Preparations of arsenic are found to be of great service in this form of eczematous inflammation. A form of arsenical remedy known as the Asiatic pill is recommended by Cazenave. Its composition is as follows:—

℞. Arsenici Protoxidi, gr. i.; Pip. Nig., gr. xii.; Pulv. Acac., gr. ii.; Aq. Distill., q. s.—Divide in pil. xii. vel. xvi. One pill to be taken once, twice, or thrice daily after meals.

But it is of the greatest importance that the meals and the diet should be regulated. Small doses of *pilulæ hydrargyri,* or of *pilulæ calomelanos composita,* or *hydrargyra c. cretâ,* may be given at bedtime for a few days, and followed each morning by a *drachm of magnesia,* given in combination with a teaspoonful of *lemon juice.* The vascular excitement of the stomach is best subdued by dilute hydrocyanic acid, in the following formula (A. T. Thomson):—

℞. Potassæ Carbonatis, ʒss.; Succi Limonis Recentis, *f* ʒiv.; Acid. Hydrocyanici Dilutis, ♏iv.; Vini Seminum Colchici, ♏xv.; Aquæ Distillatæ, *f* ʒvi.; *misce. Fiat haustus* 4tâ *quâque horæ sumendus.*

The tone of the stomach is then to be restored by small doses of *bicarbonate of potassa,* with from twelve to fifteen minims of the *tincture of henbane,* in a fluid ounce and a half of the *infusion of cinchona.* The eruption and itching disappear as the mucous membrane of the stomach and bowels return to a healthy state. The diet should be absolutely free from stimulants; and tepid baths, at a temperature of 96° Fahr., used every morning for half an hour, are most serviceable. The bowels should be regularly relieved by such mild measures as the following formula for pills:—

℞. Pilulæ Hydrargyri, gr. vi.; Pulveris Ipecacuanhæ, gr. vi.; Extracti Colocynthidis Comp., gr. xii.; Ext. Hyoscyami, gr. xviii.; *misce. Fiat pilulæ duodecem. Sum.* ij. h. s. *quotidie* (A. T. Thomson).

Glycerine and emollient lotions are useful local applications. I have often seen the itchiness greatly relieved by the following lotion of bitter almonds rubbed night and morning over the parts affected:—

℞. Amygdal. Amar. Numero, xx.; Aquæ Rosæ, ʒvii. *Contunde et tere simul, dein cola, et adde* Hyd. Corrosiv. Sublim., gr. xii.; Ammoniæ Hydrochloratis, gr. xii.; Spt. Vini Rect., ʒi.

PHLEGMONOUS INFLAMMATIONS.

Of the phlegmonous inflammations of the skin, erysipelas may be considered the type. Its pathology has been already described (vol. i., p. 354).

NEW FORMATIONS.

Warty Disease of the Skin.—Ichthyosis involves the whole tissue of the skin, and is characterized by the growth of thick, dry, imbricated scales of a dirty grey colour, resting upon a perfectly uninflamed surface, and never accompanied by pain, heat, or itching. It appears principally on the external parts of the limbs, round the joints, on the knee and elbow, on the upper part of the back, and generally on those regions where the skin is thick and coarse. The disease is generally congenital, and lasts during life. It may not be strongly marked at the period of birth; but the skin appears dull, thick, and fretted, and the disease is developed as the infant grows older.

Treatment.—Remedial measures are only palliative, consisting mainly of mucilaginous and glycerine lotions, with vapour baths to molify the roughness. Potash solutions or other preparations of potash are contra-indicated as local applications.

Cheloid, or *Keloid.*—A tuberculated growth or swelling of the true skin, originally described by Alibert, subsequently by the late Dr. Addison, of Guy's Hospital, and Mr. Thomas Longmore, Professor of Military Surgery at the Army Medical School.

The keloid of Alibert is a fibrous tumor of the skin, often developed on a cicatrix; whereas true keloid, as described by Dr. Addison and Mr. Longmore, is a much more extensive lesion. The true keloid begins as a few prominent red tubercles, generally over the sternum—whence it may extend gradually to the sides of the body and the back. The red tubercles gradually increase in size, and coalesce into larger growths. These growths then send forth spur-like processes on every side; a slight puckering of the healthy skin surrounds the marginal limits of the bases of the tumors, and marks the advance of the disease. The spread of the disease is very slow and gradual, extending over months and even years. Intense itching and irritation accompany its advance, more particularly when warm in bed at night, or during hot weather. Continued cold causes the growths partially to disap-

pear, leaving an old cicatrix-like condition of the skin (*Med.-Chir. Trans.*, 1863).

Treatment is not yet defined; but the history of the disease points to cold applications and tonic remedies as the basis of a rational treatment.

Molluscum.—This disease consists of numerous small tubercles, varying in size from that of a pea to a pigeon's egg. They are round, flat, and irregular in shape, and sometimes seated on a broad base, or attached to a peduncle. They grow very slowly, and may last through life, occurring chiefly on the face and neck, but may cover the whole body. Writers are not agreed as to the true nature of this disease. A contagious variety has been described by Bateman, Carswell, Dr. John Thomson, and Dr. Robert Paterson, of Leith, whose excellent and erudite paper, in the *Edinburgh Medical and Surgical Journal*, No. 148, will well repay a careful study.

Treatment.—The tubercles of molluscum seem to diminish under the use of arsenic. Sulphate of copper, solid or in strong solution, effects the destruction of the tumors with sufficient rapidity; but caustic potass is by far the most speedy and effectual method of cure (ROBERT PATERSON).

THE

SCIENCE AND PRACTICE OF MEDICINE.

PART IV.

MEDICAL GEOGRAPHY; OR, THE GEOGRAPHICAL DISTRIBUTION OF HEALTH AND DISEASE OVER THE GLOBE.*

CHAPTER I.

SCOPE AND AIM OF THIS BRANCH OF SCIENCE.

THIS department of the Science of Medicine treats of the manner, and endeavours to investigate the conditions under which diseases are distributed over the world, or are confined to certain districts. It embraces a consideration of topics which constitute the basis of

* Our knowledge on this subject is as yet only beginning to assume a shape; and the limits of this text-book merely permit the most faint outline to be given. To Alexander Keith Johnston, F.R.S.E., the medical profession in this country is indebted for bringing the subject prominently forward, in a communication to the Epidemiological Society of London, published in their *Transactions* for 1856, p. 25, and also in his *Physical Atlas of Natural Phenomena*, where his observations at p. 117 are illustrated by a map. "No scholar out of the domain of medicine has contributed documents more valuable than these to medical literature." That map Mr. Johnston has reduced to a scale suited to this handbook, and thus liberally permits me to use it in illustration. The most important works or monographs which have been published on this subject are Mühry's *Outlines of Noso-Geography*, in two volumes; and Boudin's *Traité de Géographie et de Statistique Médicales, et des Maladies Endémiques*, Paris, 1857, 2 vols., 8vo. A paper on Acclimation by Dr. J. C. Nott, in a work entitled *The Indigenous Races of the Earth;* Sir Alexander Tulloch's *Army Statistics;* Marshall's "Sketch of the Geographical Distribution of Diseases," in the *Edinburgh Medical and Surgical Journal*, vol. xxxviii., p. 330, and vol. xliv., p. 28; Dr. A. S. Thomson's *Thesis on the Influence of Climate on the Health and Mortality of the Inhabitants of the different Regions of the Globe*, Edinburgh, 1837; and Sir Ranald Martin's work on the *Influence of Tropical Climate*, are the sources from which the outline here given has been compiled.

Hygiology, and which are of the greatest importance to Practical Medicine, of the utmost interest to Science, and of inestimable value in Political Economy. It embraces the medical application of the facts of *physical geography,* combined with those of *vital statistics;* and it has been variously named MEDICAL GEOGRAPHY or NOSO-GEOGRAPHY.

Geographical Distribution of Disease-Realms.—As the physiological conditions of plants and animals vary according to different degrees of latitude, or rather with the different lines of equal temperature and moisture north and south of the equator, so do the pathological characters of diseases differ; and races of men are influenced as to health in proportion as they migrate from the land of their birth. It is the ascertained facts in meteorology and climatology of our globe which will help to explain the geographical limits of particular diseases, and their regulated distribution according to atmospheric temperature and moisture, the density and electricity of the air, and the vegetation with which they are surrounded. Such causes determine some of the laws by which diseases may be geographically distributed; but other concurrent causes must also be taken into account in considering the special diseases of countries. For example, topographical situation, geological nature and elevation of the soil, and state of the vegetation; in short, *physical climate,* generally and properly so called, combined with the habits of the people, their attention to personal hygiene and general sanitary arrangements, all concur to stamp the diseases of certain countries with a special character, and facilitate or retard their propagation.

There are facts which show that certain diseases are so completely under the influence of temperature that they are susceptible of being arranged systematically in zones of geographical distribution. The diseases susceptible of being thus classified are those of a communicable type, which require a certain range of temperature and concurrence of physical conditions for their prevalence and propagation. They are chiefly *yellow fever, plague, typhus fever, typhoid fever,* and *cholera.* The geographical distribution of these diseases into zones, north and south of the equator, appears to be regulated, in a great measure, by relative degrees of temperature and humidity in the several places where they prevail, in America, Asia, Africa, and Europe. Such diseases as have been mentioned, and whose realms are bounded in a

great measure by isothermal lines, are not only associated with locality, with characteristic vegetation, with heat and humidity, but they also follow the physiological habits of the several animal inhabitants of the different latitudes. *Malarious fevers, yellow fever, plague, typhus,* and *typhoid fevers,* have particular climates or zones where each predominates, and beyond the limits of which the disease is rarely if ever perpetuated when imported, unless change of seasons gives rise to a state of climate analogous to that in which the specific disease is known to flourish.

Isothermic Zones bear a most prominent part in relation to the geographical distribution of diseases. They connect the different places on the earth which have the same mean temperature, and which Humboldt was the first to indicate. *Insect-realms* have been similarly indicated by Latreille. The late Professor Edward Forbes described *homozoic belts* of marine life. Cuvier, Blumenbach, Morton, Latham, Prichard, and others, have indicated *homoicephalic zones* or *realms of men;* and, following out these ideas, we have realms of disease defined by Dr. Mühry and Mr. Keith Johnston.

Realms of Disease and Description of the Map.—These zones, belts, or realms of particular types of disease, thus marked out on the globe by these observers, are intimately associated with temperature, and may be generally indicated by the regions of the *tropical, temperate,* and *polar* zones, distinguished on the map by the respective colours of *brown, green,* and *blue.*

I. The northern limit of the tropical zone unites with the southern limits of the temperate zones, and the lines of union of the two colours on the map (green and brown) indicate Humboldt's mean annual isothermal line of 77° Fahr. or 19° Reaumur. It passes through Cuba and Florida in America; skirts the Cape de Verd Islands to Africa, where, extending beyond the usual limits of the tropics, it passes the northern part of the great desert (Sahara) below Algiers, runs through Egypt, Northern Arabia, and Persia, into China, where it is lost in the Pacific Ocean, below the limits of the northern tropic. The limiting line of this zone ascends somewhat in summer, when the sun is north of the equator; and descends again in winter, when the sun is to the south of it. To the south of the equator the same *isothermal line* (77°) marks the southern limits of the tropical *disease-realm,* where it joins with the northern limits of the *south*

temperate zone. It crosses South America near the Amazon district, and approaching southwards in Africa towards the Cape, crosses over and embraces the northern half of Australia. This is the *realm* of tropical diseases, and is coloured *brown* on the map. The class of diseases which characterize this realm are the worst forms of *malarious* (*intermittent* and *remittent*) *fevers*, associated more especially with *dysentery, diarrhœa, malignant cholera, specific yellow fever, hepatic affections, and their results.* Our summer and autumnal affections, characterized by *biliousness, diarrhœa, and bilious, gastric, or typhoid fevers*, approach, by the phenomena they express, the type of the *tropical* diseases. The paludal fevers of this tropical *disease-realm* prevail in their greatest intensity in flat, low-lying countries in the vicinity of marshes, the borders of lakes, shores of rivers and of the sea, and especially where the soil is damp underneath, and of certain geological formation. Sir Ranald Martin has clearly shown how the various soils affect powerfully the temperature and humidity of a place. Argillaceous and ferruginous soils appear in this realm to be especially insalubrious. The malarious fevers of this region make their appearance soon after the setting in of the rainy season, or when overflowed grounds, such as rice-fields, the partially dried-up beds and mouths of rivers, or irrigated plains, begin to dry up and leave portions of the surface of the land, whose subsoil is constantly wet, exposed to the rays of a tropical sun. From such a surface the belief is now universal that a miasm (of the specific nature of which we know nothing) emanates, and acts as a poison upon the blood; and is apt to be developed under similarity of climate, season, and soil, and to produce diseases whose symptoms and course express a constancy and similarity of type. The great centres of these malarious diseases in the different continents are,—(1.) In America—the shores of the Gulf of Mexico, the West India Islands, and the northern portion of South America; (2.) In Asia—India, China, Borneo, Ceylon; (3.) In Africa—the countries round the Gulf of Guinea on the west, Madagascar and Mozambique on the east, Algeria and the shores and islands of the Mediterranean on the north.

The whole mortality within this realm of disease amongst native troops (as estimated by Mr. Johnston) is about 18 per 1,000 for all India, and about 75 per 1,000 for the more unhealthy

districts. An average mortality may therefore be stated of about 46 per 1,000 for the realm of disease now under consideration, or 4·6 *per cent.* annually. Active disease and premature death have been such common occurrences in India, that people have been led to believe these results to be the price which we must pay for the tenure of that country. Because disease and premature death were so common, they ceased to arrest public attention, and the causes of them, so frequently pointed out by medical authorities in India, were generally disbelieved by the home authorities when reported to them. At last the *Report of the Royal Commission on the Sanitary State of the Army in India* has disclosed *to the public* the real state of the case, so long known to the intelligent but helpless medical officer. The state of affairs had been unceasingly represented by such men as Jackson, Sir Ranald Martin, Norman Cheevers, Morehead, W. C. Maclean, Macpherson, and many others. It has been promised by the Home Government that the sanitary reforms and improvements indicated by the Commission in their published Report shall be carried out. Several great measures advocated in the Report, and most ably urged by Miss Nightingale in her excellent pamphlet, *How to Live and not Die in India,* have already been carried out. A Commission of Health has been appointed for each Presidency, and that for Bengal has already given public evidence of its zeal. These Commissions have been put into communication with the Barrack and Hospital Improvement Commission at the War Office, which now contains members representing the Indian Government; and by this time the Indian Commissions at the several Presidencies are in possession of all the more recent results of sanitary works and measures which have been of use at home. The condition of the soldier in India is being improved. The worst personal causes of ill-health to which he was in former times exposed have been, or are being, removed. The soldiers are allowed and encouraged to cultivate gardens and to work at trades. The regulation *two drams* of alcoholic spirits have been reduced to one, and that *one dram* is to be diluted with water. "But the main causes of disease in India—want of drainage, want of water-supply for stations and towns, want of proper barracks and hospitals—remain as before in all their primitive perfection."

The maximum intensity of *dysentery, yellow fever, diarrhœa,*

malarial fevers, and *affections of the liver*, is observed in those countries which are situated under the line of greatest annual mean temperature—namely, 82° Fahr., which is the assumed equator of heat of the globe. This line of greatest heat also intersects the region of the earth where the greatest amount of water is deposited. Northward to 23° of north latitude, 53 *per cent.* of all the deaths are attributable to these diseases. In latitude 35° north, where the greatest annual temperature is only 77° Fahr., and just where the zone of the tropical disease-realm merges into that of the temperate realm of disease, these diseases cause about 14 *per cent.* of the whole mortality. To the southward, again, in the same latitude—namely, 35°, at the Cape of Good Hope—these diseases yield a loss of only 3 *per cent.* of the whole deaths.

II. The next well-marked *realm* is that in which varied forms of *continued febrile* disease take the place of the *malarious* or *paludal* fever of the torrid zone. The region where diseases of this type prevail embraces *realms* to the north and south of the equator which may be generally described as in the north and south temperate zones. The southern boundary of the northern realm corresponds to the northern boundary of the tropical *disease-realm*, as already indicated by the isothermal line of 77°. In a northern direction it extends to latitude 60° north, and includes the British Isles, Norway, and Sweden; while in America its northern limit includes part of Nova Scotia and Newfoundland. Its boundary line to the north passes from 60° north latitude in a south-eastern direction on both continents, till it gradually declines towards the borders of Asiatic Russia in the Old World, and to the district between Boston and Philadelphia on the eastern shores of the New, and corresponds nearly with the annual isotherm of 41°. In the southern hemisphere it embraces the southern two-thirds of South America, the district of the Cape of Good Hope, and the southern half of Australia. It embraces the most healthy regions of the world, in which the prevailing causes of ill-health are mostly due to the *condensation of people in towns*, and the insalubrious and depressing conditions which necessarily arise from that cause. Emanations from nuisances tend to accumulate, where mechanical and chemical arrangements of a sanitary kind do not remove the concentrated impurities.

But where proper sanitary measures exist for the supply of pure water and free air, the artificial disadvantages inseparable from town life may be greatly modified in their influences, so much so, that in our country (the least unhealthy of all) the mortality may range from 1·7 to 3·6 *per cent.* annually (Reg.-Gen. *Rep.* for 1853, p. 15). Nearly every type of disease has a representative in this realm; but, generally speaking, the general and mixed diseases are the classes which furnish the greatest mortality. *Typhus* and *typhoid fever* prevail between the parallels of 44° and 60° in Western Europe; *yellow fever* has prevailed on the southern shores of Spain, the north-west coast of France, and Northern Italy; *intermittent fever* in the Netherlands, Sweden, and Central Italy, and generally where marshy, undrained lands exist. *Small-pox* especially prevails where vaccination has not checked its ravages; *leprosy* and *elephantiasis* prevail in Scandinavia; *pellagra* in Italy, France, and Spain; *plica polonica* in Poland and Tartary; and *consumption* and *rheumatism* everywhere. While, therefore, this temperate zone embraces, on the one hand, the extremes of temperature of the torrid and the frigid zones in the seasons of summer and winter, it is also observed to have representatives of the types of disease which prevail in both these realms; and according as we approach its northern or its southern limits we find that the characters of disease become so modified and their types are found so to mingle together on the confines of these disease-realms that the *continued* type of febrile diseases peculiar to the temperate zones tend, as we approach the tropics, to merge into and to participate in those irregular intermittent characters which are peculiar to the type of the tropical malarious districts. It is only to a limited extent, however, that such modifications are found to occur; and there are some diseases which rarely tend to pass their geographical boundaries unless especially favoured by *tropical* identity of climate on the one hand, or *temperate* identity on the other. Under the former circumstance yellow fever has been met with at Gibraltar, Cadiz, Lisbon, St. Nazaire, and even so far north as Plymouth Sound and Southampton Water; and it has also extended in America southwards beyond its usual limits; but so soon as the temperature falls below 55° Fahr., the importation of yellow fever into *this zone,* and its propagation or persistent existence in it, becomes impossible. Diarrhœa, also, and dysentery are apt to prevail

during the summer and autumn months; while under bad diet, defective ventilation, and generally imperfect sanitary measures, *contagious typhus fever* is apt to be propagated in crowded localities of towns, and in huts, hospitals, and barracks. Typhus and typhoid fevers seem to have their special habitation in this zone, especially between 30° and 40° of north latitude, and likewise the true glandular plague; and which may be occasionally propagated beyond these limits by a secondary and specific poison, generated from human bodies and propagated by human intercourse. The zone, therefore, which we inhabit, while it may be said to exhibit the greatest variety of disease-processes and types of disease, may, nevertheless, be considered a highly favoured district of the globe, inasmuch as the development and progress of disease and of epidemics are much more manageable, less intense, and on the whole less fatal, than similar types of disease in the tropical realm, or even than they were centuries ago. Under proper sanitary regulations *typhus fever* and many other diseases in this country are greatly under control. *Cholera* may be said to be the only scourge which occasionally, as an epidemic, becomes *rapidly* destructive to life. But, by scientifically directed sanitary measures, and the increasing experience of an enlightened age, the spread of the *plague* in this country has ceased. In this realm "history records the existence of diseases of former days now happily unknown; while, on the other hand, scourges unknown to ancient times devastate modern populations" (PLINY, SYDENHAM, BOUDIN). In the sixteenth century the Oriental plague, "like the destroying angel, spread its wings on the blast," and added to the miseries of that stormy and bloody time. Our physician-poet tells us that when the fate of England was about to be decided on "Bosworth's purple field," that plague, of most gigantic arm,

> "Rushed as a storm o'er half the astonished isle,
> And strewed with sudden carcases the land."

Then, as now, our standing armies and bodies of men congregated together especially suffered from pestilential disease. It so thinned the ranks of Henry's victorious army that few were left to see the conqueror crowned with Richard's diadem. Asiatic cholera may now be said to have taken the place of the Oriental plague, and it is often no less destructive and important in its results—sternly demonstrating "how one dread year

performs the work of ages, when the pestilence mocks in his fury the slow hand of time."

III. To the northward of this temperate zone, in the northern hemisphere, there is another disease-realm, where *catarrhal affections, influenza, scurvy, erysipelas, diseases of the skin* and *digestive organs,* and *various* CONSTITUTIONAL *affections* more especially prevail, to the exclusion of *malarious febrile diseases,* except on very rare occasions in summer, and when *cholera* and *dysentery* may prevail. This *disease-realm,* in the polar isothermal zone, rejoices in a climate directly opposite to that of the tropical zone. Its southern limits are the northern boundaries of the previously defined region—namely, the isothermal line of 41° Fahr., or 2° or 3° of Reaumur. Commencing on the western coasts of North America above Sitka, it extends southward across the district of the Canadian lakes, sinking south and east into Canada and Newfoundland to Boston and New York or Philadelphia. Thence it continues north-west nearly on 41° Fahr. annual temperature, when it crosses to Europe, and ascends till near the borders of Iceland, whence it sinks towards Norway and Sweden, and, running above St. Petersburg and Moscow, crosses to Siberia. Iceland being the best-known locality of this district, Mr. Johnston takes its peculiar diseases as the representatives of this realm. Every year, in spring or in early summer, it is visited by *catarrh;* and at short intervals it is visited by *catarrhal fever*—a *true influenza,* which has usually a great effect on the mortality. The majority of Icelanders are said to die before the age of fifty, from *asthmatic* or *catarrhal affections,* which are also prevalent in Greenland and Labrador.

Dr. Lawson has attempted to establish (see vol. i., p. 223) the occurrence between 1817 and 1836 of a series of *oscillations of febrile diseases,* following each other over the world with amazing regularity. These he attributes to a cause or influence which, from its extent and progressive character, he names a "pandemic wave," to distinguish the influence from that usually understood as "epidemic," referring to a single form of disease, affecting a limited space. Under the influence of this pandemic wave he believes that there is a constant progressive tendency to the development of various endemic febrile diseases in the Atlantic

and western parts of the Indian Ocean, from south or south-east to north or north-west.

Many of the facts and data on which he founds are, however, not sufficiently trustworthy to rest a judgment upon, and in some cases are susceptible of a totally different interpretation from that which Dr. Lawson has assigned to them. Although, therefore, his theory is "not proven," yet the expression of it is calculated to do good, by drawing attention to this view of the subject.

CHAPTER II.

ON MALARIA AND PLACES KNOWN AS MALARIOUS.

GEOGRAPHICAL facts, collected by medical writers from Hippocrates downwards, show that every country is unhealthy in proportion to the quantity of marsh, or of undrained alluvial soil, it contains, the inhabitants of such districts dying often in the ratio of 1 in 20, instead of 1 in 38—the average mortality in healthy countries. The connection of a given class of disease—represented by remittent and intermittent fever—with marshy districts is now distinctly established and generally recognized.

Ancient Rome was once the seat of so many fatal epidemics that the Romans erected a temple to the goddess Febris. These epidemics were known to arise from the great masses of water poured down from the Palatine, Aventine, and Tarpeian hills becoming stagnant in the plains below, and converting them into swamps and marshes. The elder Tarquin ordered them to be drained, and led their waters by means of sewers to the Tiber. These subterraneous conduits ramified in every direction under the city, and were of such considerable height and breadth that Pliny terms them "*operum omnium dictu maximum suffossis montibus atque urbe pensili subterque navigata.*" This system of drainage, which was continued as late as the Cæsars, rendered Rome proportionably healthy, and the seat of a larger population than has since perhaps been collected within the walls of any city. On the invasion

of the Goths, however, the public buildings were destroyed, the embankments of the Tiber broken down, the aqueducts laid in ruins, the sewers obstructed and filled up, and the whole country being now again overflowed, Rome has once more become the seat of an almost annual paludal fever, as in the times of her earliest foundation. The insalubrity of the Pontine Marshes, past or present, is notorious. Three hundred years, however, before the Christian era, Appius Claudius drained them, by making canals, building bridges, and by constructing that magnificent road, portions of which still remain, and still bear his name. On the invasion of Italy by Theodoric, Cæcilius Decius gave a free course to the waters in the neighbourhood of Rome, and the re-establishment of these immense marshes was one of the many disasters which resulted from the attacks of the Goths on Italy. Their present state is such that the Tuscan portion of Maremme, and indeed the whole of that district, may be said in summer to be absolutely depopulated, not a single house retaining an inhabitant, except the guard-houses, with a few soldiers and custom-house officers; and these are relieved twice or thrice during the summer, with the Maremme fever almost invariably upon them.

Many districts in the East and West Indies, in Pennsylvania, and Continental Europe, are known to be active in the evolution of *malarious influences.* Such places are generally the deltas, marshy banks, and embouchures of rivers, in the plains extending from the bases of mountain ranges; partially inundated and irrigated lands, or such as are traversed by percolating streams or canals in wooded districts termed *jungles;* the seaboard, especially where there is jungle or salt marsh; and in the Bengal district the stations of Calcutta, Chinsurah, and Berhampore are highly malarious (MARTIN). The woods and marshes of the Sunderbunds, covering a superficies of more than 20,000 miles, and extending 180 miles south and east of Calcutta, composed of marshy land, covered with forest and underwood, together with the numerous embouchures of the Ganges, are well-known unhealthy districts. The partially dried-up marshes and beds of rivers have too often been fatal to our armies when imprudently and ignorantly encamped in their vicinity. In 1810 the plains of Spain, along the course of the Guadiano, with its "lines of detached pools" and its ravines always "half-dried,"

could tell of a fever-stricken army. The pages of history also remind us how our British soldiers perished on the low, dry-looking, sandy plains of Walcheren and of Rosendaal in 1794 and 1809-10. Our last war with Russia, during the campaign in Bulgaria, and especially at Varna in 1854, furnishes a no less melancholy record of the sufferings of British troops, and the persistent pernicious influence of a residence in malarious districts.

In China we know of the miasmatic nature of the deltas of the Blue and the Yellow Rivers. In Africa we know of the Zais, the Orange, and the Zambesa as unhealthy rivers. In America the Amazon, the Orinoco, and the Rio-del-Norte are similarly deleterious. In England we have the fens of Norfolk and Lincolnshire still a source of disease; in short, there is hardly a country which has not its marshy lands, so that abundance of work exists for chief commissioners of sewers to direct for good, where such "heads" of "*bodies*" exist; and the extent of disease proceeding from marshes has been shown in many places in Italy, Sicily, and Greece to be so great as to occasion more than two-thirds of the average mortality.

Removal and Neutralization of Malaria.—Of towns that have been drained and remain healthy there are many examples in ancient and modern history. Hippocrates tells us that the city of Abydos had been several times depopulated by fever; but the adjoining marshes having been drained, it became healthy. London, in the time of Sydenham, was infested with epidemic intermittent fever and dysentery, the mortality from the former alone averaging, in a comparatively small population, from one to two thousand persons annually. In the present day, owing to the formation of sewers and a general system of drainage, a case of ague contracted in London is hardly known. Many other towns, both of this country and of France, as Portsmouth, Rochefort, and Bordeaux, from being the constant seat of paludal fevers, have been, from the same causes, rendered in like manner comparatively healthy. Dr. Wood, of Pennsylvania, relates an interesting fact regarding the neutralization of miasmatic effluvia. He tells us they are in some way rendered innocuous by the air of large cities. This fact is notorious in relation to the city of Rome; and it is abundantly confirmed in the larger towns of the United States, in the neighbourhood of which these diseases have prevailed.

Nature of the Noxious Agent.—Thus the intimate connection between marshy districts and certain forms of disease is established by a great amount of direct and indirect testimony; but two questions still remain—namely, What is the nature of the noxious agent of malarious districts? and what circumstances are necessary to its formation or extrication? It seems certain that the deleterious agent is neither heat alone nor moisture alone, nor any known gas extricated from the marsh. It cannot be *heat* alone, for many of the hottest parts of the West Indies are free from fever. It cannot be *moisture* alone, for no persons enjoy better health than the crews of clean ships at sea, even when cruising in tropical climates, as long as they have no communication with the land. While *carbonic acid, azote, oxygen,* or *carbureted hydrogen,* the gases collected by stirring the bottom of marshes, have all been inspired without producing any disease similar to paludal fever, it seems to follow almost as a necessary consequence that the remote cause must be a *miasm, poison,* or *malaria,* whose presence is solely detected by its action on the human body; and two hypotheses have been imagined to account for its origin;—the one, that it is a product of vegetable decomposition—the other, that it is an exhalation from the earth, favoured by the conditions of marshiness. A theory, often hinted at, is beginning to find expression—namely, that very minute fungi of rapid growth, like *smut, rust, mildew,* and the like, are active agents in the propagation of malaria. Inspector-General Reid has shown this in regard to the recent epidemic in the Mauritius, and Dr. Massey in regard to Ceylon (see *Army Med. Report* for 1867).

The general evidence in favour of vegetable decomposition (and therefore growth) being the remote cause is, that all countries are for the most part free from paludal diseases while the crops are growing, and only become unhealthy after the harvest, when large quantities of vegetable matters are left on the ground at the time the rain begins to fall. Marshes are in general healthy till the summer's sun, or other cause, has diminished their waters, and bared a greater or less portion of their bed. The part thus exposed almost always contains a large portion of vegetable matters, which, running into rapid decomposition, generates other vegetable growths of a fungoid nature especially, which may be or may convey the poison which gives origin to this class of disease.

It is during the periods of the year when the drying process is in greatest activity that unhealthiness prevails with greatest severity in the East Indies—namely, before the commencement and after the termination of the rainy season. The particular evidence of vegetable decomposition being the source of the poison is of the following nature:—Lancisi gives the history of an epidemic which for several summers infested, and almost depopulated, the ancient town of Urbs Vetus, situated on an elevated and salubrious part of Etruria, and which was traced to the circumstance of the peasants steeping their flax in some stagnant water in the neighbourhood of the town. This practice was therefore prohibited in 1705, and the epidemic ceased to appear. The steeping of flax being productive of paludal fever is a fact the knowledge of which is not limited to Italy; for the ancient as well as the new "*coutumes*" of almost all the provinces of France have proscribed the steeping of flax, "la rouissage," even in running waters, from the fear of infection. In the Netherlands the same belief has prevailed. In July, 1627, the King of Spain passed an ordinance prohibiting the steeping of flax in the streams and canals of Flanders. The prohibition may be explained on another ground —namely, because the flax poisons the water and kills the fish. In Ireland, and perhaps also elsewhere, steeping flax in a running stream is therefore forbidden by law.

The experience of the indigo-planter is to the same effect. In India, after the colouring matter has been extracted from the indigo plant, it was formerly the custom to throw the detritus into large heaps or masses in the immediate neighbourhood of the works, and which, at the end of three or four years, becomes manure of an excellent quality. It was found, however, that these heaps, wetted from time to time by the heavy rains, and afterwards heated by the rays of a burning sun, rapidly decomposed, and at length emitted *miasmata*, which produced all the effects of those extricated from the marsh. The workmen who lived near, and more especially those to leeward of these masses, were found to be very commonly attacked by fever, chiefly of the remittent type, and similar to those which prevail in the paludal districts of that country. This consequence is now so well established that the most intelligent indigo-planters no longer allow these heaps to be formed either near the works or in the immediate neighbourhood of the cottages of their workmen.

Ships also afford additional evidence of the truth of the hypothesis of vegetable decomposition and growth being the remote cause of malaria.

All intertropical regions, where the nature of the locality admits only of a rice cultivation, are well known to be unhealthy.

These facts render it highly probable that the noxious agent must be a product of vegetable development, growth, and propagation, evolved on the soil, and moving in the lower regions of the atmosphere. No analysis of the air has yet disclosed any immediate chemical principle to which the unhealthy influence of miasms may be ascribed. The atmospheric air collected at the embouchure of the Valtelline—a country where it is impossible to sleep without being attacked with fever—gives, on analysis, the same chemical constituent parts and proportions of gases as that collected at the summit of the Alps, or in the narrowest streets in London. But such places, on the other hand, give evidence of abundant minute fungoid vegetation, which may be active poisons, or active agents in propagation of malaria.

If we consider the paludal poison to be a product of vegetable decomposition, or of vegetable growth, it follows that heat and moisture, quantity of vegetable matter, and nature of the soil, though not the essential agents, must have a sensible influence on its formation, must vary its intensity or quantity, and also must limit paludal diseases to particular localities, seasons, and latitudes. A certain temperature, for example, under certain conditions as to moisture, is evidently necessary to its extrication and development.

It is certain also that a given quantity of moisture is as necessary to vegetable decomposition or growth as a given temperature, and that the extrication of the paludal poison will be most abundant from that soil which contains no more moisture than is necessary for that process; for an excess in quantity, by dividing and separating the particles, and by preventing the access of atmospheric air, will either retard or altogether put a stop to putrescency. Hence in some countries frequent and heavy rains will render marsh fevers prevalent, by saturating the whole of the open country; while privation of rain will in others produce exactly the same effect in other instances, merely by diminishing the superfluous quantity of water. Thus, in the West Indies, an uncommonly rainy season seldom fails, in the perfectly dry and

well-cleared Island of Barbadoes, to induce for a time general sickness; while at Trinidad, whose central portions are described as a sea of swamp, and where it rains nine months in the year, an excess of moisture is a preservative from sickness; for should at any time rains fall only eight months in the year instead of nine, the swamps become dry and bared to the sun, and remittent fevers of the worst kind are sure to make their appearance. The same result follows on the subsiding of the waters of rivers that have overflowed their banks, as those of the Nile, the Rhone, the Danube, the Tigris, the Ganges, and many others.

It is evident from these data that the swamp, on its approach to dryness, is the source of disease and death; while an excess of rain has a preservative power so long as moisture is in excess. On the contrary, on the rich and dry plains, and even on the hills of tropical countries, rain is the cause not only of vegetable decomposition, but also of disease; while absence of rain tends to preserve health.

In estimating, however, the dryness of a country, its superficial appearance is often deceitful. In the years 1748 and 1794 the summers were dry, and our troops took up the encampments of Rosendaal and Ousterhout in South Holland. The soil in both places is a level plain of sand, with a perfectly dry surface; and where no other vegetation existed, or could exist, but a few stunted heath-plants; yet in both years fever became epidemic among the troops in each place. On digging for water the cause was discovered, for the soil was found to be saturated with water to within a few inches of the surface. It is probable, therefore, that this country was originally formed of vegetable and other detritus, brought down by the Rhine and the Waall, and afterwards covered with sand thrown up by the sea, and which, heated by the summer's sun, became the powerful cause of the extrication of marsh miasmata. From the exceeding malignity of the salt marshes, it has been supposed that a mixture of salt and fresh water renders a marsh more pernicious than either of them alone. It has been found, however, that on coasts where these marshes have been kept up to one uniform level by means of flood-gates, the surrounding country is healthy; and it has therefore been inferred that the sickness produced is a consequence of the perpetual alteration of the level of the waters of the marsh, and not owing to the admixture of sea and spring water.

It is probably owing to a great excess of temperature that rocky countries, as Gibraltar and the Ionian Islands, are so often and so severely attacked with malarious fever. It is on the summits of these rocks that springs arise. The slightest frost produces fissures, into which fungi, as "*mould*," and other vegetable matters insinuate themselves, while the bare rock becomes heated to an intense degree. Humboldt, on ascending the Orinoco, found the station at the great fall depopulated by fever, which the natives attributed to the bare rocks of the rapids. He determined the heat of these rocks to be 118·4° Fahr., while the temperature of the air immediately around was only 78·8° Fahr. Again, the rock of Gibraltar is known to be percolated with water, so that we can hardly conceive a more pestilential focus of disease when the causes necessary to the formation of miasm or fungi are combined. The existence of paludal fever in dry and rocky districts, therefore, although it may appear extraordinary and unexpected, is not necessarily an exception to the general law of paludal diseases being generated by miasmata, the result of vegetable decomposition or of vegetable development. In many hot climates the most deadly sites for encampments have been the dried-up beds of rivers, or their immediate vicinities (MARTIN).

These facts seem, therefore, unquestionably to prove that heat and moisture, though not the primary cause of paludal disease, are conditions essentially connected with the development of vegetable growths, like fungi, of the noxious miasmata, and consequently furnish a strong additional argument in favour of the hypothesis of vegetable decomposition generating the remote cause which produces or propagates some miasmatic diseases. It is certain, however, even when the conditions of heat, moisture, and vegetable matter most abound, that paludal diseases do not always assume their severest forms: and there seems reason to believe that differences of geological formation, by favouring or otherwise influencing vegetable putrefaction and growth, may variously affect the health of countries similarly situated in other respects.

It is perfectly well known that different soils radiate heat with different degrees of intensity, and consequently are, under the same circumstances, of different temperatures, having very different powers of attracting moisture; and possibly, also, they may have other and more direct properties favourable to the

generation of the paludal miasm. Nothing, for instance, is better determined in husbandry than that the *carbonate of lime*, mixed with the ordinary matters of a compost, greatly forwards the processes of putrefaction, so that the mass thus prepared is fit in a much shorter time for the purposes of manure.

There are some soils peculiarly favourable to the decomposition and growth of vegetable matters, and consequently to the more abundant extrication of marsh miasmata; and it is remarkable that those countries most celebrated for paludal fevers have been found similar in their geological formation to each other, and to those artificial conditions which most favour rapid vegetable decomposition and growth.

To predicate all the facts connected with paludal diseases is not yet possible; for the variations of atmospheric temperature, the changes in the quantity and nature of the electric fluid, the quantity of water, the nature of the soil, the amount and character of the vegetable matters and their growth, form a problem extremely complicated, and one whose smallest variation as to quantity or time may occasion marked differences in the result. As a general rule, however, it may be stated, that in no climate do paludal fevers prevail to an equal degree all the year round. In the winter, much of the vegetable matter has already undergone decomposition and further growth, while the dryness of the season and the diminished temperature are little favourable to its further development. When the spring, however, arrives, and the rain falls, and the heat of the sun increases, the earth again evolves a miasm of mitigated intensity. In summer the products of vegetable decomposition are used up in affording nourishment to the growth of many vegetable forms; and this season, like the winter, is in general healthy. But in the autumn, and after the harvest has been gathered, when the ground is covered with vegetable debris, when the rain falls in torrents now and again, and when the solar heat has acquired its greatest intensity, all the conditions for the greatest amount of growth of vegetable matter, of moderate moisture, and of highest temperature, change and are united; so that the season which realizes the hopes of the husbandman is also the period of pestilence and of his greatest danger. There are two other facts, also, which are too prominent to be mistaken. The one is, that the miasmata vary greatly in intensity in different countries, and also in different parts of the

same country: again, the diseases they produce, though annually *endemic* in given districts, yet become, in certain years, and from the action of causes not yet determined, *epidemic*.

In the same countries, also, it is determined that difference of altitude is equivalent to difference of latitude; and, as a general law, it may be stated that in the Antilles, on the continent of America, from Boston to Rio de Janeiro, and also on the continents of Asia and Africa, while in the low country severe remittent or yellow fever prevails, still in the higher country, though immediately contiguous, the type is changed to intermittent and mild remittent. The interesting fact stated by Humboldt, that the *vomito prieto* never appears on the table-lands of Mexico, is strictly in accordance with the observations made in every other equatorial part of the world at a similar elevation above the level of the sea. The circumstance of intermittents passing into remittents, and remittents into malarious yellow fever, and, conversely, of remitting and malarious yellow fever often terminating in intermittent—facts observed not only in the East and West Indies, but on the continents of America and of Africa—demonstrates a unity of cause as firmly as the best-established facts in medicine.

That paludal diseases, like many diseases produced by morbid poisons, are annually endemic, and only occasionally epidemic, is unquestionable. A few years ago intermittent fever was epidemic in particular districts in this country; but of late years the cases of ague have been comparatively rare. In Demerara it has been observed that malarious yellow fever is epidemic about every seventh year. At Gibraltar, although sporadic cases of paludal fever occur annually, still malarious yellow fever is only occasionally epidemic, but so irregularly that it assumed that character in 1804, then in 1810, again in 1813 and in 1814, and from that period the garrison suffered no similar visitation till 1828. The physical causes on which this greater virulence and greater spread of the disease depend are not determined. In temperate climates it has been observed that paludal fevers have been most prevalent when a hot summer has succeeded a wet spring.

Infecting Distance of Miasmata.—As a general law, the danger of infection is in proportion to the proximity to a marsh. But there are many disturbing causes which produce remarkable exceptions to this law. These disturbing causes are, the extent of surface

which generates the miasmata, their intensity, the direction of the wind, its force, the season of the year, the time of the day, and the attracting influence of the surface over which the miasmata pass.

The Altitudinal Range.—The Monte Mario, which adjoins Rome, is, according to Breyslack, about 165 yards perpendicular height above the Pontine Marshes, and is extremely unhealthy. Trivoli, which is about 230 yards above the level of the same marshes, is infinitely more salubrious; while at Serre, 340 yards perpendicular height, the inhabitants enjoy an entire exemption from the paludal diseases which prevail below. In Italy it is estimated that an altitude of 1,400 to 1,600 feet is necessary to assure an exemption from paludal disease; but in the West Indies, where the poison is of so much greater intensity than in Italy, it is estimated that an elevation of 2,000 to 2,500 feet is necessary to give a similar immunity. The different latitudes may account for this.

In towns partially freed from marsh miasmata by extensive drainage, the difference of a few feet perpendicular height makes an almost inconceivable difference in the liability of persons to paludal disease. The barracks of Spanish Town, the capital of Jamaica, for instance, consist of two storeys, or of a ground floor and of a first floor; but it being found that two men were taken ill on the ground floor for one on the first floor, it was at length ordered that the ground floor should be no longer occupied. Dr. Cullen remarked a similar result at Portobello, Dr. Fergusson in St. Domingo, and Sir Gilbert Blane in the expedition to Walcheren. This law is so well understood in the West Indies that in Demerara, and in many other parts, the houses are built on dwarf columns, after the manner of corn stacks, in order that a stratum of air may be interposed between the house and the ground. In Rome, and in other towns of Italy, it is also so well known that the lower rooms of the houses are abandoned, the family occupying the upper rooms, as affording a greater protection from the paludal poison.

The Lateral or Horizontal Spread of marsh miasmata is a problem still more difficult than that of the altitudinal range. The least complicated cases are those when water alone intervenes between the marsh and the recipient. In the year 1746-47, while our troops lay in Zealand, the sickness was so great among four bat-

talions quartered there that some of those corps had hardly 100 men fit for duty, or less than a seventh part of a battalion. In one corps, the Royals, only four men escaped. At the time, however, of this remarkable prevalence of fever on shore, Commodore Mitchell's squadron lay at anchor between South Beveland and the Island of Walcheren, and the fever raged at both places; but, nevertheless, in the midst of all the sickness that reigned around, the seamen were neither affected with fever nor flux, but continued to enjoy perfect health. These observations of Sir John Pringle were fully confirmed by those of Sir Gilbert Blane during the last disastrous expedition to Walcheren. "I had," says this physician, "the opportunity of observing the extent to which this noxious exhalation extended, which was found to be less than was generally known. Not only the crews of the ships in the Road of Flushing were entirely free from this epidemic, but also the crew of the guard-ship, which was stationed in the narrow channel between this island and Beveland. The width of this channel is about 6,000 feet; yet, though some of the ships lay nearer to one shore than the other, there was no instance of any of the men or officers being taken ill with the same disorder as that with which the troops on shore were affected." It appears, therefore, that in Europe the horizontal spread of marsh miasmata over fresh water is less than 3,000 feet. With respect to the spread of the miasmata over salt water, Sir Gilbert Blane wrote that in tropical climates ships at a distance of 3,000 feet from a swampy shore—a distance to which the miasmata did not extend in Zealand—and even farther, were affected with the noxious exhalations. Dr. John Hunter considered a few miles to be a necessary interval for a ship lying to leeward of a swamp, in order to insure a complete exemption from the disease. When, however, the swamp or other source of the poison is of small extent, a much less space is sufficient to assure an exemption. In the epidemic on the coast of Spain, the fisherman living with his family on board his boat has been rarely attacked, though lying at anchor close in shore. Also, during the late epidemics at Gibraltar, it was not unusual for the richer inhabitants to hire a Moorish vessel and to live on board in the bay; and there was scarcely an instance of those persons having been affected, though keeping up a free communication during the day, either directly or indirectly, with the town.

The extent to which the marsh miasmata may spread from its source over land in a horizontal direction is a much more complicated question. The effect of trees in intercepting the paludal poison is remarkable, and appears to have been known to the ancients, who are supposed to have surrounded their temples with groves, on account of their protecting influence. Pope Benedict XIV. ordered a wood to be cut down which separated Villatri from the Pontine Marshes, and for many following years there raged throughout the whole country, and in places never before attacked, a most severe and fatal fever. The same effects were produced from a similar circumstance in the environs of Campo Santo. In the West Indies it is quite wonderful how near the marsh the planter, provided he is protected by trees, will venture to place his habitation.

Different soils also affect the transmission of the paludal poison. The spot, for instance, on which the new National Dock and Arsenal are built was a marsh of about 700 acres, and on either side of it are the villages of Greenhithe and of Northfleet. The peculiarity here is, that the inhabitants of these villages rarely suffer from intermittent fever, whilst those on the hills beyond are greatly afflicted with that disease. Dr. Maton mentions a similar fact in the neighbourhood of Weymouth, and the same circumstance is observed in the neighbourhood of Little Hampton and the marshy districts in Sussex.

CHAPTER III.

ACCLIMATION, OR THE INFLUENCE OF CLIMATE ON MAN.

Definition.—*Acclimation consists in "a profound change in the organism, produced by a prolonged sojourn in a place whose climate is widely different from that to which one is accustomed, and which has the effect of rendering the individual who has been subjected to it similar in many respects to the natives of the country* (indigènes) *which he has adopted"* (Rochoux). *The process takes place to a certain extent so far as some individuals are concerned; but the ability to become acclimated is not possessed to the same extent by all nations.*

It is found that the *white races* reach their highest physical and intellectual development, as well as most perfect health and greatest average duration of life, above 40° in the western, and 45° in the eastern hemispheres; and whenever they emigrate many degrees below these lines they begin to deteriorate, from increased temperature, either alone or combined with other morbific influences, incident not less to change of climate than to habits of life—to evils social, civil, and more or less remediable by attending to the common principles of sanitary science.

The laws of climate show that each race of mankind has its prescribed salubrious limits. All of them seem to possess a certain degree of constitutional pliability, by which they are able to bear to a certain extent great changes of temperature and latitude; and those races that are indigenous to temperate climates support best the extremes of other latitudes. The inhabitants of the arctic regions, and of the tropics, have a certain pliancy of constitution; but while the inhabitants of the middle latitudes may emigrate 30° south or 30° north with comparative impunity, the Esquimaux in the one extreme, or the Negro, Hindoo, or Malay in the other, have no power to withstand the vicissitudes of climate encountered in traversing the 70° of latitude between Greenland and the equator. The fair races of Northern Europe below the arctic zone find Jamaica, Louisiana, and India, to be extreme climates: and they and their descendants are no longer to be recognized after a prolonged residence there. When an Englishman is placed in the most beautiful part of Bengal or Jamaica, where malaria does not exist, although he may be subjected to no attack of acute disease, but may live with a tolerable degree of health his threescore years and ten, he nevertheless ceases to be the same healthy individual he once was; and, moreover, his descendants degenerate. He complains bitterly of the heat, and becomes tanned; his plump, plethoric frame becomes attenuated; his blood loses fibrine and red globules; both mind and body become sluggish; grey hairs and other marks show that age has come on prematurely—the man of forty looks fifty years old; the average duration of life is shortened (as shown in life insurance tables); and the race in time would be exterminated if cut off from fresh supplies of emigrants from the home country. The European

in the Antilles *struggles with existence*—a prey to fever and dysentery. He is unequal to all labour, becomes wasted and wan, and finally perishes. His decay is premature; and, but for the constant influx of fresh European blood, he becomes rapidly extinct as a race. The European inhabitants of Jamaica, of Cuba, of Hispaniola, of the Windward and Leeward Islands, have made no progress since their first establishment there. They cannot execute labour—hence the necessity for preserving and maintaining the black population. Their offspring are pale, wan, and sickly, and in half a century cease to be productive (MORELL, p. 107). Our army medical historians tell us that our troops *do not become* acclimated in India. Length of residence in a distant land affords no immunity from the diseases of its climate, which act with redoubled energy on the stranger from the temperate zones. On the contrary, the mortality among officers and troops is greatest among those who remain longest in those climates (JOHNSON, MARTIN, TULLOCH, MACPHERSON, BOUDIN). Dr. Macpherson also makes the significant remark, that the small mortality among officers, compared with soldiers, in India is due to the greater facilities they enjoy of obtaining change of climate when they fall sick. Although the constitution of the man may be so modified that comparative health may be retained, yet there is a *morbid degradation* of the physical and intellectual constitution. If, however, he or his descendants are taken back to their native climate, they may yet revert to the healthful standard of their original types. The good effects of limiting the period of service of our troops in the West Indies to three years has shown this, in sustaining for a greater period the strength of the regiments, a protracted residence of the European regiments in India having been followed by the most disastrous results. "European regiments in India have melted away like the spectres of a dream. A thousand strong men form this year a regiment: a year passes, and *one hundred and twenty-five* new recruits are required to fill up the broken column; and, eight years having come and gone, not a man of the original thousand remains in the dissolving corps." With regard to the Bombay fusilier European regiment, for instance, Dr. Arnot has shown that its losses averaged 104 per 1,000 per annum—a loss equivalent to the entire absorption of the regiment in nine years and seven months. In Bengal, also, it is an ascertained fact that a

British regiment of 1,000 men dissolved entirely away in eleven years, even in favourable times, and with all the improved conditions of the service. Dr. Arnot's statistics show that the Bengal army lost annually 9 *per cent.* of its numbers, giving a total loss in eight years of upwards of 14,000 men out of an army of 156,130 men. (See *Trans. Med. Ph. Society of Bombay* for 1855; *Indian Annals of Medical Science;* and *Sanitary Review* for October, 1857; whence these statements are quoted.)

In the *Statistical Reports of the Army Medical Department,* of which four annual volumes have now been published, for the years 1859 to 1862 inclusive, full information respecting the health of the army all over the world has been given by Dr. T. Graham Balfour, F.R.S., Deputy Inspector-General of Hospitals, to whom the science of medicine is so largely indebted in the direction of "Medical Geography." The leading facts are embraced in the following account of—

The Sickness and Mortality of British Troops at Different Places over the Globe.

I. **In the Stations of the United Kingdom** the sickness is represented by 1,025 admissions, and the mortality by 9·35 deaths *per* 1,000 men.

The class of diseases which gives rise to the largest proportion of cases is that of *Venereal,* by which one-third of all the admissions into hospital is caused. Next to it rank *miasmatic diseases, diseases of the integumentary system,* and *diseases of the respiratory system. Tubercular diseases,* chiefly *phthisis,* occasion upwards of one-third of the whole mortality, and diseases of the *respiratory system* stand next to them. The admissions by *tubercular diseases* average 19 *per* 1,000, and the deaths 3·48; the admissions by diseases of the *respiratory system* amount to 98 *per* 1,000, and the deaths to 1·47 *per* 1,000.

II. **The Mediterranean Stations.**—1. *Gibraltar.* The sickness here is represented by 894 admissions, and the mortality by 8·86 deaths *per* 1,000 men. Of *miasmatic diseases* the *continued fevers* are the most prevalent, and at the same time the most fatal. The sickness from them is represented by 94 admissions, and the

mortality by 2·62 deaths *per* 1,000 men. The causes of such prevalence are represented to be—(1.) Overcrowding—the space in one of the barracks having been, during part of the time included in the Reports, only 322 cubic feet per man! (2.) Exposure to sun-heat; (3.) Imperfect drainage and sewerage. *Diarrhœa, dysentery,* and *cholera* are FOUR TIMES as prevalent as among troops at home, and occasion 73 admissions, *rheumatism* 38 admissions, and *ophthalmia* 41 admissions *per* 1,000 men.

Venereal diseases give rise to nearly one-fourth of the admissions into hospital.

2. *Malta.* The sickness at this station is represented by 906 admissions, and the mortality by 12·31 deaths *per* 1,000 of mean strength.

The diseases of the *miasmatic class,* in the order of their greatest prevalence, are as follow:—

Continued fever, represented by 188; *ophthalmia,* by 81; *dysentery* and *diarrhœa,* by 73 admissions *per* 1,000 men. Of these diseases *continued fever* has been the most fatal.

3. The sickness in the Ionian Islands is represented by 807 admissions, and the mortality by 9·11 deaths *per* 1,000 of mean strength.

The most prevalent diseases are *continued fever, dysentery* and *diarrhœa,* and *ophthalmia.* The *continued fevers* are the fatal ones. Their prevalence and fatal character in Corfu are attributed, and apparently with justice, to the unhealthy situation of the Fort-Neuf Barracks, their overcrowded state and defective ventilation, and to the generally insanitary condition of the town.

III. **The Stations in British America.**—1. In *Bermuda* the sickness is represented by 637, and the mortality by 11·79 deaths *per* 1,000 of mean strength. *Continued fevers, dysentery,* and *diarrhœa* are the most prevalent diseases, and those of the nervous system the most fatal, owing principally to the number of deaths from *delirium tremens.* Bermuda has been visited at irregular intervals by yellow fever in an epidemic form, but no cases of it have occurred since the commencement of the publication of the Army Annual Reports. In the year 1864 a deadly epidemic prevailed.

2. In *Nova Scotia* and *New Brunswick* the sickness is repre-

sented by 581 admissions *per* 1,000 of mean strength, and the mortality by 7·43 deaths *per* 1,000. The most prevalent diseases of the miasmatic class are *sore throat, influenza,* and *rheumatism.* The tubercular diseases are the most fatal, causing nearly one-third of the whole mortality. Next to them stand diseases of the respiratory organs—chiefly *pneumonia* and *bronchitis.*

3. In *Canada* the sickness is represented by 622 admissions *per* 1,000 of mean strength, and the mortality by 9·07 deaths *per* 1,000. The miasmatic diseases, in the order of their greatest prevalence, are as follow:—*Dysentery, diarrhœa, cholera,* represented by 28 admissions *per* 1,000 of mean strength; *rheumatism,* by 28 *per* 1,000; *sore throat* and *influenza,* by 26 *per* 1,000; *continued fevers,* by 20 *per* 1,000; *ophthalmia,* by 16 *per* 1,000. Accidents, tubercular diseases, and those of the respiratory system, are the chief causes of mortality; miasmatic diseases (chiefly *continued fevers*), and those of the nervous system, being next to them.

4. In *Newfoundland* the sickness is represented by 980 admissions, and the mortality by 6·72 deaths *per* 1,000 of mean strength. The most prevalent diseases are *sore throat, influenza, bronchitis,* and *rheumatism.*

5. In *British Columbia* the sickness is represented by 702 admissions *per* 1,000 mean strength, and the mortality by 14·52 deaths *per* 1,000; but of the eight deaths which occurred during four years, five were by drowning, and one that of a man frozen to death; so that two only, or in the ratio of 0·36 *per* 1,000 of mean strength, were the result of disease. *Influenza* and *venereal* are the prevailing diseases.

IV. **West Indian Stations.**—1. Among **White Troops** of the *Windward* and *Leeward Command* the sickness is represented by 1,112 admissions *per* 1,000 of mean strength, and the mortality by 10·91 deaths *per* 1,000. The *miasmatic* diseases, arranged in the order of their prevalence, are as follow:—*Paroxysmal fevers,* represented by 282 admissions; *ophthalmia,* by 116; *dysentery, diarrhœa, cholera,* by 46; *continued fevers,* by 39; and *rheumatism,* by 26 admissions *per* 1,000 of mean strength. *Yellow fever* sometimes prevails. *British Guiana* is the most sickly station; *Trinidad* the most deadly.

Among **Black Troops** the sickness is represented by 915 admissions, and the mortality by 20·89 deaths *per* 1,000 of mean

strength. *Miasmatic* and *enthetic* diseases are the most prevalent, and *tubercular* and *miasmatic* the most fatal. The *miasmatic* diseases, arranged in the order of their prevalence, are as follow:—*Paroxysmal fevers*, represented by 94 admissions *per* 1,000 of mean strength; *rheumatism*, by 86; *eruptive fevers*, by 28; *sore throat* and *influenza*, by 27; *ophthalmia*, by 25; and *dysentery, diarrhœa*, and *cholera*, by 18 admissions *per* 1,000. *Tubercular diseases* are most fatal to this class of troops, causing nearly one-third of all the deaths; next to these rank *diseases of the respiratory system, diseases of the nervous system, paroxysmal fevers*, and *dysentery* and *diarrhœa*.

2. The sickness in *Jamaica* among **White Troops** is represented by 896 admissions *per* 1,000 mean strength, and the mortality by 14·08 deaths. The most prevalent of the *miasmatic* diseases are as follow:—*Ophthalmia*, represented by 117 admissions *per* 1,000 mean strength; *continued fever*, by 74; *paroxysmal fevers*, by 63; *dysentery, diarrhœa*, and *cholera*, by 42; *rheumatism*, by 18; *sore throat* and *influenza*, by 9 admissions *per* 1,000.

The most fatal diseases are *paroxysmal* and *continued fevers*.

Among the **Black Troops** in *Jamaica* the sickness is represented by 1,090 admissions, and the mortality by 28·61 deaths *per* 1,000 of mean strength.

The most prevalent *miasmatic* diseases are *paroxysmal fevers*, represented by 277 admissions *per* 1,000 of mean strength; *rheumatism*, by 64; *dysentery, diarrhœa*, and *cholera*, by 24; *continued fever*, by 22; *ophthalmia*, by 21; *sore throat* and *influenza*, by 18 admissions *per* 1,000.

Tubercular diseases and those of the respiratory system are the most fatal, causing upwards of two-fifths of all the deaths. Next to them are paroxysmal fevers, and diseases of the digestive and nervous systems.

3. In the *Bahamas*, among **Black Troops**, the sickness is represented by 841 admissions, and the mortality represented by 29·08 deaths *per* 1,000 of mean strength.

Miasmatic diseases are the most prevalent, and tubercular the most fatal. The mortality is also high from those of the respiratory system.

4. In *Honduras*, among the **Black Troops**, the sickness is represented by 1,164 admissions, and the mortality by 18·23 deaths *per* 1,000 of mean strength.

The prevalent diseases are *miasmatic.* Skin diseases also abound, especially *eczema, ulcers,* and *boils.*

V. **West African Stations.**—1. In *Sierra Leone* the sickness is represented by 740 admissions, and the mortality by 29·53 deaths *per* 1,000 of mean strength.

2. At the *Gambia* the sickness is represented by 978 admissions, and the mortality by 33·74 deaths *per* 1,000 of mean strength.

3. On the *Gold Coast* the sickness is represented by 624 admissions, and the mortality by 26·45 deaths *per* 1,000 of mean strength.

The *Gambia* and *Sierra Leone* seem to be the most sickly; but the average duration of the cases of sickness, and the mortality compared with the admissions, is greatest on the *Gold Coast.*

The most prevalent miasmatic diseases in *Sierra Leone* are as follow:—

Paroxysmal fevers, represented by 98 admissions *per* 1,000 mean strength; *rheumatism,* by 56 *per* 1,000; *dysentery* and *diarrhœa,* by 20; *eruptive fevers,* by 23 *per* 1,000.

The most prevalent on the *Gambia* are,—

Paroxysmal fevers, represented by 294 admissions *per* 1,000 of mean strength; *dysentery, diarrhœa,* and *cholera,* by 58 *per* 1,000; *eruptive fevers,* by 44 *per* 1,000; *rheumatism,* by 28 *per* 1,000; *ophthalmia,* by 13 admissions *per* 1,000 men.

The most prevalent on the *Gold Coast* are,—

Dysentery, diarrhœa, and *cholera,* represented by 31 admissions *per* 1,000 mean strength; *rheumatism,* by 26 *per* 1,000; *ophthalmia,* by 19 *per* 1,000; *paroxysmal fever,* by 11 admissions *per* 1,000. *Small-pox* and *measles* are sometimes epidemic; and *Guinea-worm,* among parasitic diseases, holds a prominent place. Indeed, in 1861, this affection was the cause of one-third of the admissions into hospital; and it is worthy of remark that in that year not a single case occurred among the troops at Sierra Leone and the Gambia.

VI. **St. Helena.**—The sickness is represented by 880 admissions *per* 1,000 mean strength; the mortality, by 11·28 deaths *per* 1,000.

The prevalent diseases are *dysentery* and *diarrhœa,* represented by 104; *continued fever,* by 124; *ophthalmia,* by 27;

paroxysmal fever, by 32; *rheumatism*, by 26; *sore throat* and *influenza*, by 13 admissions *per* 1,000 of mean strength.

The most fatal diseases are those of the nervous system, tubercular diseases, and *continued fevers*. *Intemperance* prevails to a great extent.

VII. **Cape of Good Hope.**—The sickness is represented by 873 admissions *per* 1,000 mean strength; the mortality, by 10·87 deaths *per* 1,000.

The most prevalent miasmatic diseases are *ophthalmia*, represented by 91 admissions *per* 1,000 mean strength; *continued fever*, by 49; *rheumatism*, by 41; *dysentery*, *diarrhœa*, and *cholera*, by 45; *sore throat* and *influenza*, by 20; *paroxysmal fevers*, by 9 admissions *per* 1,000.

The most fatal of these diseases is *continued fever*. Intemperance prevails to a large extent, and is among the chief causes of the prevalence of diseases of the heart in this station (NICHOLSON, TAYLOR). *Ocular* and *cardiac diseases* are the cause of the discharge of half the invalids from the Cape. The mortality by diseases of the circulatory system amounts to 1·58 *per* 1,000 of mean strength annually.

VIII. **Island of Mauritius.**—The sickness is represented by 915 admissions, and the mortality by 25·04 deaths *per* 1,000 of mean strength. This ratio of mortality, however, may be considered as above the usual average, having been raised by the prevalence of a very fatal epidemic of cholera in 1862.—On the average of the three preceding years it only amounted to 17·39 *per* 1,000.

The prevalent diseases are *dysentery* and *diarrhœa*, represented by 179 admissions *per* 1,000 men; *continued fevers*, by 68; *spasmodic cholera*, by 25; *rheumatism*, by 25; *ophthalmia*, by 19; *sore throat* and *influenza*, by 10 admissions *per* 1,000 men.

The most fatal diseases are *spasmodic cholera*, *dysentery* and *diarrhœa*, and *continued fevers*.

IX. **Ceylon.**—The sickness among **White Troops** is represented by 1,513 admissions, and the mortality by 23·55 deaths *per* 1,000 of mean strength.

The most prevalent diseases of the miasmatic class are *dysentery*, *diarrhœa*, and *cholera*, represented by 222 admissions *per* 1,000 men; *paroxysmal fever*, by 121; *ophthalmia*, by 107; *rheumatism*, by 75; *continued fever*, by 44.

The fatal diseases are *dysentery, diarrhœa,* and *cholera;* diseases of the digestive system, particularly *hepatitis, tubercular diseases,* and those of the *nervous system.*

Among the **Black Troops** the sickness is represented by 1,064 admissions, and the mortality by 11·97 deaths *per* 1,000 of mean strength.

The prevalent diseases are *paroxysmal fever,* represented by 391 admissions; *continued fever,* by 60; *ophthalmia,* by 75; *rheumatism,* by 46; *dysentery, diarrhœa,* and *cholera,* by 56; *eruptive fevers,* by 10 admissions *per* 1,000 men.

The fatal diseases are *spasmodic cholera, dysentery* and *diarrhœa,* and *paroxysmal fevers.*

Trincomalee is the most sickly station. Ague prevails there from the marshy soil of *paddy* fields and cocoa-nut gardens.

X. **Australasia.**—1. *Australia and Tasmania.* The sickness is represented by 726 admissions *per* 1,000 of mean strength, with a mortality of 15·51 deaths *per* 1,000.

The miasmatic diseases, in the order of their greatest prevalence, are *dysentery, diarrhœa,* and *cholera,* represented by 87 admissions *per* 1,000; *rheumatism,* by 51; *continued fever,* by 21; *sore throat* and *influenza,* by 50; *ophthalmia,* by 47 admissions *per* 1,000 men.

The most fatal diseases in this command are the *tubercular,* which caused 4·67 deaths per 1,000 of strength. Diseases of the *circulatory system* gave rise to 2·34; and those of the *nervous system* to 2·13. Of the miasmatic diseases, *continued fever* is the most fatal, the deaths by it amounting to 1·06 per 1,000 of the strength.

2. *New Zealand.* The sickness is represented by 595 admissions *per* 1,000, and the mortality by 13·25 deaths. But of these 4·16 were the result of wounds in action, leaving a proportion of only 9·09 *per* 1,000 resulting from the ordinary contingencies of colonial service.

The miasmatic diseases, in the order of greatest prevalence, are *ophthalmia,* represented by 50 admissions *per* 1,000 men; *dysentery* and *diarrhœa,* represented by 44; *rheumatism,* by 37 admissions *per* 1,000; *sore throat* and *influenza,* by 36; *continued fever,* by 17; *paroxysmal fever,* by 8 admissions *per* 1,000 men.

The most fatal diseases are those of the *tubercular class. Con-*

tinued fever gave rise to only ·53 deaths *per* 1,000 of the strength; and *dysentery* and *diarrhœa* caused exactly the same proportion.

XI. **China.**—The sickness among **European Troops** is represented by 1,968 admissions, and the mortality by 57·81 deaths *per* 1,000. The sickness among **Native Troops** is represented by 1,724 admissions, and the mortality by 33·53 *per* 1,000.

1. *In Southern China* the sickness among **European Troops** is represented by 2,340 admissions *per* 1,000, with a mortality represented by 35·49 *per* 1,000.

The miasmatic diseases, in the order of greatest prevalence, are as follow:—*Paroxysmal fevers*, represented by 571 admissions *per* 1,000; *continued fever*, by 236; *dysentery, diarrhœa*, by 211; *ophthalmia*, by 109; *rheumatism*, by 75; *sore throat* and *influenza*, by 16 admissions *per* 1,000 men.

The fatal diseases were *dysentery* and *diarrhœa, paroxysmal continued fevers*, and *spasmodic cholera*.

The sickness amongst **Native Troops** in *Southern China* is represented by 1,286 *per* 1,000, and the mortality is represented by 30 *per* 1,000. The diseases arranged in the order of greatest prevalence are as follow:—*Paroxysmal fevers*, represented by 603 admissions *per* 1,000 men; *dysentery, diarrhœa*, and *cholera*, by 166; *rheumatism*, by 89; *continued fever*, by 25; *sore throat* and *influenza*, by 13; *ophthalmia*, by 12 admissions *per* 1,000 men.

The fatal diseases were *dysentery, diarrhœa, cholera, paroxysmal continued fever*, and *rheumatism*.

In *North China* the results are so much affected by the peculiar circumstances of field service in the campaign of 1861, and the military operations around Shanghai in 1862, that they would only mislead if stated as the effect of climate. In 1861, at Tein-tsin, 19 deaths were recorded from *sunstroke* between the 17th and 23rd of July, the *maximum* temperature during that period ranging from 95° to 108° Fahr. in the shade—giving a maximum average of 104° 6′; and the minimum temperature ranged from 73° to 83° Fahr. in the shade; the average being 79° 2′ (Dr. Lamprey). Information on this important point is exceedingly meagre. The experience of Surgeon Butler at Meean Meer led him to write that "when the thermometer ranges beyond 98° Fahr., in crowded barracks, cases of heat apoplexy almost invariably occur." (See p. 380, *ante*.)

In 1862 cholera broke out in the field force in the neighbourhood of Shanghai, and cut off 58 *per* 1,000 of the strength. It also attacked the small garrison left at Taku, and the mortality in it amounted to 40 *per* 1,000.

XII. **Stations in India.**—The prevalence of sickness, on the average of the three years 1860-62, as shown in the *Army Medical Department Reports*, is represented by 1,818 admissions *per* 1,000 mean strength, and the mortality is represented by 32·45 deaths. The relative sickness and mortality are distributed over the three Presidencies as follow:—

The sickness in *Bengal* is represented by 1,944 admissions, and the mortality by 37·43 deaths *per* 1,000; the sickness in *Madras*, by 1,342 admissions, and the mortality by 19·82 deaths *per* 1,000; the sickness in *Bombay*, by 1,844 admissions, and the mortality by 27·15 deaths *per* 1,000.

In all three Presidencies *paroxysmal fevers* form the group of miasmatic diseases which gives rise to the highest proportion of admissions; but in Madras these are scarcely one-third as prevalent as in Bengal and Bombay. *Continued fevers* are very common in Bengal, and *dysentery and diarrhœa* are the cause of the admission into hospital of between 150 and 160 *per* 1,000 of the strength in each of the Presidencies. *Spasmodic cholera* has not only been the cause of the highest rate of mortality in all three Presidencies, but has been a source of much greater mortality in Bengal than in the others. Next to it come *dysentery* and *diarrhœa*, and after them *paroxysmal fevers*.

The lives wasted in India in each year of service (according to the Commissioners' Report) appear to have been greater in the Queen's regiments than in the Company's regiments. Half of the deaths take place during the first five years; and the numbers invalided increase slightly in each quinquennial period. The decrement year by year of the force in India has been such that 1,000 effectives are reduced to 96 men in 20 years (that is, by death alone, to 344—by death and invaliding, to 216—by death, invaliding, and other causes, to 96). The mean term of service in India has been 8·6 years, and 11 recruits are required annually to every 100 men; and to maintain 85,856 men 10,000 annual recruits are required; and these are reduced to less than half their original numbers in *eight* years. The half of a regular

army so constituted consists of men who have served less than six years; and not more than a fourth of the men are veterans of ten years' standing, on whom the discipline and solidity of an army greatly depend.

The Endemic Diseases of India in the order of greatest prevalence are *paroxysmal fevers, continued fevers, dysentery, diarrhœa, rheumatism, ophthalmia, spasmodic cholera, sore throat,* and *influenza.* Arranged in the order of comparative mortality, the diseases are as follow:—*Spasmodic cholera, dysentery, diarrhœa,* and *continued* and *paroxysmal fevers.* These are especially the diseases of the sultry plains of India.

The Fevers are especially the paludal fevers; but both typhus and typhoid fevers are now known to occur in India.

The Dysentery of India is most prevalent in the plains during the hot and rainy seasons; and amongst British soldiers it is computed that eleven cases of dysentery occur to one amongst the native soldiery. The cases of dysentery present "a spectacle of distress of as pitiable a kind as can be found in the history of human suffering."

Diseases of the Liver.—No statistics can give any idea of the extent of these diseases; and in acute inflammation, so prevalent in the plains, the danger to life is imminent from the first, and, in the event of recovery, impairment of health more or less permanent is certain.

Cholera is the most acute of acute diseases. Troops, both European and Native, while on the march, are more liable to seizure by this disease; and when the attacks have occurred in cantonments after the march, the prevalence of the epidemic is generally in proportion to the length of the march.

The annual rate of mortality among soldiers is 10 *per* 1,000 in England; in India, according to the *Commissioners' Report,* it is 67 *per* 1,000 (Bengal), of which 58 *per* 1,000 is due to zymotic diseases, the fevers killing 17 men in 1,000; dysentery and liver disease, 20; cholera and diarrhœa, 18 men in 1,000. *Delirium tremens, catarrh, syphilis, rheumatism,* and *scurvy* are much more fatal in India than in England.

Nearly all the diseases fatal in India are accompanied by profuse discharges, with which the air, water, linen, bedding, closets, walls of hospitals, and barracks become more or less infected; so that the *materies morbi* come into contact with all

the inmates of buildings where the disease prevails. In India the soldier's sickness is doubled. As to how far this sickness and mortality are aggravated by the unsanitary state of the towns and the mode of life in India, the reader is referred to the *Report of the Royal Commission on the Sanitary State of the Army in India*, and to the second edition of Miss Nightingale's pamphlet, entitled *How to Live and not Die in India.*

As legitimate deductions from the preceding statements, the following summaries of results are here given:—

(A.)

Stations of the British Army, arranged in the Order of the Greatest Number of Annual Admissions per 1,000 of Mean Strength.	Annual Admissions per 1,000 Mean Strength.	Annual Mortality per 1,000 Mean Strength.
White Troops.		
1. South China,	2,340	35·49
2. Bengal,	1,944	37·43
3. Bombay,	1,844	27·15
4. Ceylon,	1,513	23·53
5. Madras,	1,342	19·82
6. Windward and Leeward Command,	1,112	10·91
7. United Kingdom,	1,025	9·35
8. Newfoundland,	980	6·72
9. Mauritius,	915	25·04
10. Malta,	906	12·31
11. Jamaica,	896	14·08
12. Gibraltar,	894	8·86
13. St. Helena,	880	11·28
14. Cape of Good Hope,	873	10·87
15. Ionian Islands,	807	9·11
16. Australia,	726	15·51
17. Bermuda,	637	11·79
18. Canada,	622	9·07
19. New Zealand,	595	13·25
20. Nova Scotia, &c.,	581	7·13
Colonial Corps.		
1. South China,*	1,820	34·83
2. Honduras,†	1,164	18·23
3. Jamaica,†	1,090	28·61
4. Ceylon,‡	1,064	11·79
5. Gambia,†	978	33·74
6. Windward and Leeward Command,†	915	20·89
7. Bahamas,†	841	29·08
8. Sierra Leone,†	740	29·43
9. Gold Coast,§	624	26·45

* Asiatics.

† Africans and Coloured Creoles.

‡ Cingalese and Natives of India.

§ Africans.

(B.)

Stations of the British Army, arranged in the Order of the Greatest Annual Mortality per 1,000 of Mean Strength.	Annual Mortality per 1,000 Mean Strength.	Annual Admissions per 1,000 Mean Strength.
European Troops.		
1. Bengal,	37·43	1,944
2. South China,	35·49	2,340
3. Bombay,	27·15	1,844
4. Mauritius,	25·04	915
5. Ceylon,	23·53	1,513
6. Madras,	19·82	1,342
7. Australia,	15·51	726
8. Jamaica,	14·08	896
9. New Zealand,	13·25	595
10. Malta,	12·31	906
11. Bermuda,	11·79	637
12. St. Helena,	11·28	880
13. Windward and Leeward Command,	10·91	1,112
14. Cape of Good Hope,	10·87	873
15. United Kingdom,	9·35	1,025
16. Ionian Islands,	9·11	807
17. Canada,	9·07	622
18. Gibraltar,	8·86	894
19. Nova Scotia, &c.,	7·13	581
20. Newfoundland,	6·72	980
Coloured Corps.		
1. South China,*	34·83	1,820
2. Gambia,†	33·74	978
3. Sierra Leone,†	29·53	740
4. Bahamas,†	29·08	841
5. Jamaica,†	28·61	1,090
6. Gold Coast,‡	26·45	624
7. Windward and Leeward Command,†	20·89	915
8. Honduras,†	18·23	1,164
9. Ceylon,§	11·97	1,064

* Asiatics.
† Africans and Coloured Creoles.
‡ Africans.
§ Cingalese and Natives of India.

Dr. Balfour's valuable reports (at the periods of their earlier appearance) show at a glance the great diversity in the amount of sickness and mortality to which British troops were liable (in former times) at these stations, while serving in our widely extended colonial possessions; and it will be seen how great has been the reduction, especially *in the mortality* of the troops, during the four years comprised in the preceding tables, as compared with the rates when Dr. Balfour and those with whom he laboured first brought them to the notice of the Secretary of State for War. Leaving out of consideration Sierra Leone as an extreme instance of unhealthiness, and from which it has been deemed

expedient altogether to withdraw European troops, the admissions into hospital have ranged, during the twenty years subsequent to 1836, between 529 *per* 1,000 of the strength in New Zealand and 2,117 *per* 1,000 in Bombay; while the mortality has ranged between an average of 11 *per* 1,000 in Newfoundland and 76·2 *per* 1,000 in Bengal. Not only, however, do these great differences exist in the amount of sickness and mortality at different stations on the average of a series of years, but they occur to as great an extent in different years upon the same station. Thus, in the period 1817 to 1836, the sickness ranged in the Windward and Leeward Command between 1,512 and 2,365 *per* 1,000; in Jamaica, between 1,389 and 2,423; in Ceylon, between 1,216 and 2,895. At Gibraltar it ranged between 631 and 1,498; and even in Canada it varied from 847 to 1,409 *per* 1,000. But the difference in the mortality in different years is even more striking: thus, in the Windward and Leeward Command it varied between 43 and 162; in Jamaica, between 61 and 307; in Ceylon, between 34 and 218; in Gibraltar, between 8 and 128; in Canada, between 9 and 48; and in Nova Scotia, between 7 and 40 *per* 1,000. These facts strikingly illustrate the necessity for a prolonged period of observation to obtain a fair average, and for great caution in making deductions from observations extending over limited periods and insufficient numbers.

"Another very striking fact shown by Dr. Balfour's tables is the great reduction which has taken place during the last twenty years in the mortality at some of the stations—a result which seems fairly attributable, in a considerable degree at least, to the removal of those causes of disease which were brought to light by the Statistical Reports, and to the adoption of the measures then recommended for improving the health of the troops. The most striking examples of this are to be found in Jamaica, where the deaths have fallen from an average of 128 to 60·8 *per* 1,000; in Newfoundland, from 37·7 to 11; in St. Helena, from 25·4 to 12·3; and in Ceylon, from 74·9 to 38·6 *per* 1,000.

"But, with a view to judge by what measures the health of the troops was likely to be improved, it was necessary to ascertain the diseases to which the sickness and mortality were attributable. Abstracts were accordingly prepared, showing the diseases by which every admission into hospital and death had been occasioned for a period, in most of the colonies, of twenty years. These were again grouped in classes, and the results are as follow:—

"Fevers will be found the greatest cause of mortality in Jamaica, the West Indies, and Bermuda, and, prior to 1837, in Ceylon, Gibraltar, and the Ionian Islands. Diseases of the lungs have been most fatal among the troops serving at home, particularly the Foot Guards, as well as in the West Indies, Jamaica, Bermuda, Canada, and Nova

Scotia; those of the liver in Ceylon and India, and also in Mauritius and St. Helena, prior to 1837; while diseases of the stomach and bowels have been a source of considerable mortality in India, Ceylon, West Indies, Mauritius, and, prior to 1837, also at St. Helena. A comparative exemption is enjoyed by the black troops from the fatal forms of fever by which the white troops have suffered so severely; but there is a great liability among them to pulmonary disease in the West Indies and Jamaica. A comparison of the rates of mortality prior and subsequent to 1837 shows that the improvement in the health of the troops has been attributable to different causes in different colonies. Thus, in the West Indies and St. Helena it has shown itself chiefly in the reduction of deaths by *diseases of the bowels;* in Jamaica, Gibraltar, and the Ionian Isles, *by fevers;* and in Ceylon and Bermuda, by both these classes.

"The influence of age upon mortality in the army is a question of much importance with reference to the expediency of exacting from the soldier a long continuous service in our colonies, or of shortening that service, and by frequent reliefs affording him change of climate.

"The most striking feature in this particular is the rapidly progressive increase in the mortality with advancing years in tropical climates—a result completely at variance with the prevalent opinion of the advantage of long-continued residence in such climates, and of much importance in determining the question of frequent reliefs."

Although military statistics may not be proper *criteria* of the healthiness of a place, nor of the diseases affecting the civil community, yet the information is of great value, and the following tables, kindly prepared for this edition by Dr. T. G. Balfour, F.R.S., show at a glance the *sickness* and *mortality* of the British army at all the stations occupied by it, and the classes of diseases by which *sickness* and *mortality* are caused; and, if compared with previous statements, will show the great reduction which has taken place in the amount of sickness and mortality compared with former times:—

TABLES SHOWING THE PROPORTION OF ADMISSIONS INTO HOSPITAL, AND DEATHS PER 1,000 OF MEAN STRENGTH, AT EACH OF THE FOLLOWING STATIONS, ON THE AVERAGE OF THE PERIODS NOTED.

I.—European Troops.

Stations and Period of Observation.	United Kingdom, 1860-62.		Gibraltar, 1859-62.		Malta, 1859-62.		Ionian Islands, 1859-62.		Bermuda, 1859-62.		Nova Scotia, &c., 1859-62.		Canada, 1859-62.		Newfoundland, 1859-62.		Windward and Leeward Command, 1859-62.		Jamaica, 1859-62.	
	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.
Miasmatic Diseases,	223	1·06	284	4·82	420	5·98	328	4·68	188	·91	120	·69	135	1·09	376	..	536	4·45	330	6·65
Enthetic Diseases,	352	·12	213	..	111	..	120	·06	61	..	131	..	133	·05	44	..	104	..	103	..
Dietic Diseases,	7	·06	13	·18	8	·09	5	·13	26	·23	12	·57	19	·16	36	1·12	26	·20	26	·39
Parasitic Diseases,	36	·01	8	..	13	..	5	..	6	..	10	..	7	..	6	..	3	..	3	..
Diathetic Diseases,	2	·14	3	·05	3	·04	1	·19	2	·23	1	·23	2	..	7	..	6	..	2	..
Tubercular Diseases,	19	3·48	7	1·15	11	2·14	10	1·27	9	2·04	14	2·29	7	1·41	11	1·12	10	1·21	5	1·96
Diseases of the—																				
Nervous System,	20	·68	19	·23	16	·56	17	·63	23	2·94	14	·46	16	1·09	54	2·24	33	2·22	15	1·57
Circulatory System,	9	·73	6	·64	5	·51	5	·44	5	·91	3	·91	3	·73	26	..	4	·81	6	·78
Respiratory System,	98	1·47	56	·09	47	·90	46	·57	54	..	65	1·03	66	1·46	123	2·24	32	·40	48	..
Digestive System,	38	·49	38	·23	43	·60	42	·32	62	1·13	32	·34	38	·42	58	..	75	·61	92	1·17
Urinary System,	3	·10	3	·23	2	·21	2	..	1	..	3	..	2	·15	2	..	1	..	2	..
Reproductive System,	7	..	16	..	10	..	3	..	10	..	3	..	6	..	4	..	13	..	2	..
Locomotive System,	3	..	5	·09	3	..	3	·06	3	..	2	..	3	..	11	..	5	..	1	..
Integumentary System,	122	·08	137	..	136	..	138	..	103	..	73	..	85	·10	68	..	161	..	132	..
Diseases of Nutrition,	2	·03	2	..	1	..	4	..	2	..	..	..	1	..	..	..	2	..	3	..
Accidents,	80	·59	77	·41	71	1·07	74	·51	78	2·04	95	·69	97	1·88	145	..	95	·61	121	..
Battle,	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Homicide,	..	·02	..	·14	..	..	..	·06	..	..	..	·11	..	·21	..	..	..	..	..	..
Sucide,	..	·26	..	·46	..	·17	..	·13	..	1·36	..	·11	..	·26	..	..	..	·40	..	1·17
Execution,	..	·02	..	·14	..	·04	..	·06	..	..	..	..	..	·05	..	..	..	..	..	..
Corporal Punishment,	2	..	3	..	3	..	2	..	1	..	1	..	1	..	3	..	2	..	3	..
Disease not specified,	2	..	3	..	3	..	2	..	2	..	2	..	1	..	6	..	4	..	2	..
No appreciable Disease,	..	..	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..
Total,	1025	9·35	894	8·86	906	12·31	807	9·11	637	11·79	581	7·43	622	9·07	980	6·72	1112	10·91	896	14·08

I.—European Troops—*continued.*

Stations and Period of Observation.	St. Helena, 1859-62.		Cape of Good Hope, 1859-62.		Mauritius, 1859-62.		Ceylon, 1859-62.		Australia and Tasmania, 1859-62.		New Zealand, 1859-62.		South China, 1859-62.		Bengal, 1860-62.		Madras, 1860-62.		Bombay, 1860-62.	
	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.
Miasmatic Diseases,	356	2·61	283	2·17	355	17·16	587	9·15	227	2·13	205	1·06	1236	24·21	1114	24·84	568	8·12	1049	15·82
Enthetic Diseases,	97	..	182	·06	78	..	233	·27	121	..	30	..	465	..	341	·32	269	·35	317	·31
Dietic Diseases,	43	·43	11	·53	17	·14	18	·27	24	..	11	·60	30	·36	9	·05	17	·12	18	·19
Parasitic Diseases,	3	..	15	..	10	..	4	..	1	..	13	..	5	..	7	..	9	..	12	..
Diathetic Diseases,	5	·43	3	·12	4	..	4	·27	5	·42	6	·23	5	..	6	·22	5	·18	7	·19
Tubercular Diseases,	3	1·74	9	1·18	12	1·69	18	3·08	18	4·67	8	2·04	9	·95	11	2·48	16	1·88	9	1·53
Diseases of the—																				
Nervous System,	27	2·17	24	1·35	25	1·13	54	2·50	24	2·13	22	·60	32	2·37	34	2·28	33	1·59	33	1·53
Circulatory System,	8	·43	22	1·58	11	·56	17	·83	13	2·34	7	·98	13	·59	10	·62	15	1·27	12	·53
Respiratory System,	47	·87	57	·94	62	·56	86	1·10	52	1·06	64	·76	105	1·66	57	1·04	49	·59	51	·96
Digestive System,	55	·43	46	·94	89	1·41	177	3·88	46	·64	33	·68	117	2·26	140	3·85	130	3·41	122	3·64
Urinary System,	4	..	2	·18	3	..	5	·27	4	·64	2	·23	2	·12	2	·14	2	·18	3	·06
Reproductive System,	3	..	9	..	11	..	15	..	1	..	4	..	6	..	11	·02	12	..	8	..
Locomotive System,	2	..	4	..	5	..	4	·27	3	..	3	..	5	..	5	·03	5	·03	4	..
Integumentary System,	78	..	103	..	136	..	145	..	71	..	81	·23	150	·12	108	·07	107	·18	102	·06
Diseases of Nutrition,	..	..	1	..	10	·14	8	..	..	..	1	..	3	·71	8	·14	5	·21	5	..
Accidents,	143	2·17	88	1·52	75	1·27	127	1·39	99	1·06	86	1·44	94	1·66	74	·73	91	1·27	89	1·68
Battle,	..	..	..	..	..	..	..	..	..	..	10	4·16	..	..	..	..	..	..	..	..
Homicide,	..	..	..	·06	..	..	..	..	..	..	..	·08	..	·12	..	·03	..	..	..	..
Suicide,	..	..	..	·18	..	·98	..	·27	..	·21	..	..	..	·12	..	·20	..	·06	..	·34
Execution,	..	..	..	·06	..	..	..	..	..	..	..	·08	..	..	..	·01	..	..	..	·03
Corporal Punishment,	1	..	4	..	3	..	3	..	2	..	3	..	4	..	1	..	1	..	1	..
Disease not specified,	5	..	10	..	8	..	8	..	14	·21	6	·08	32	·24	6	·36	8	·38	2	·22
No appreciable Disease,	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..
Total,	880	11·28	873	10·87	915	25·04	1513	23·55	726	15·51	595	13·25	2340	35·49	1944	37·43	1342	19·82	1844	27·15

II.—Colonial Troops.

Stations and Period of Observation.	Windward & Leeward Command, 1859-62.		Jamaica, 1859-62.		Bahamas, 1859-62.		Honduras, 1859-62.		Sierra Leone, 1859-62.		Gambia, 1859-62.		Gold Coast, 1859-62.		Ceylon, 1859-62.		China, 1859-62.	
	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.
Miasmatic Diseases,	300	5·	437	6·55	388	5·54	567	3·64	227	4·92	458	4·60	103	4·96	653	6·48	1275	23·93
Enthetic Diseases,	201	..	284	1·04	172	·69	98	..	142	..	153	1·53	41	..	30	..	95	·72
Dietic Diseases,	1	..	2	..	3	..	3	2·19	1	·70	3	..	2	..	1	..	3	..
Parasitic Diseases,	1	..	2	..	5	..	12	..	19	..	14	..	223	..	69	..	43	..
Diathetic Diseases,	5	·44	6	1·04	4	·69	6	..	4	..	9	2·30	1	..	4	·50	13	·36
Tubercular Diseases,	16	5·88	15	6·20	19	10·39	5	2·19	16	7·74	14	9·20	5	3·31	3	1·	3	·72
Diseases of the—																		
Nervous System,	17	1·96	24	2·07	16	2 08	18	1·46	14	4·22	15	1·53	12	3·31	9	·83	20	·60
Circulatory System,	4	1·09	2	·69	2	..	4	2·91	1	..	2	1·53	..	·83	2	1·	2	·48
Respiratory System,	83	2·39	73	6·20	55	6·23	65	·73	67	4·92	94	8·43	29	4·13	43	·66	84	2·63
Digestive System,	37	·65	41	2·42	16	2·08	48	·73	26	4·22	28	·77	24	2·47	32	·50	65	·84
Urinary System,	2	·65	4	..	2	..	3	..	1	..	4	·77	3	..	..	..	2	..
Reproductive System,	11	..	9	..	19	..	15	..	5	..	3	..	6	..	6	..	2	·12
Locomotive System,	3	..	2	..	1	..	4	..	13	·70	8	·77	12	..	1	..	1	..
Integumentary System,	140	·22	119	·34	73	·69	198	·73	124	..	96	..	99	..	136	..	107	·24
Diseases of Nutrition,	2	·44	..	..	2	..	1	..	1	..	1	..	..	..	1	·17	31	2 99
Accidents,	73	·65	60	·69	57	..	106	1·46	56	..	57	..	56	4·96	74	·50	71	·96
Battle,	..	..	..	..	..	..	..	..	15	2·11	8	..	..	..	..	..	..	..
Homicide,	..	·65	..	·34	..	..	..	·73	..	..	1	·77	..	..	..	..	..	..
Suicide,	..	·65	..	·69	..	·69	..	·73	..	..	..	·77	..	·83	..	·33	..	..
Execution,	..	·22	..	·34	..	..	..	..	..	..	..	·77	..	1 65	..	..	..	..
Corporal Punishment,	5	..	6	..	2	..	9	..	6	..	5	..	6	..	1	..	1	..
Not specified,	14	..	4	..	5	..	2	·73	2	..	5	..	2	..	1	..	2	·24
Total,	915	20·89	1090	28·61	841	29·08	1164	18 23	740	29·53	978	33·74	624	26·45	1064	11·97	1820	34·83

The natives of this country who are now scattered throughout Hindostan and the Indian Archipelago, on both sides of Africa, a few hundred miles north of the Cape, along the southern shores of the Mediterranean, in the West Indies, South America, and elsewhere, also illustrate the necessity of a change of climate by their habits. Few of them ever return to their native land with constitutions unimpaired; and in no cases do the British natives, whose means are not absolutely insignificant, attempt to rear up their children in any of those tropical regions. If they do so, "parents soon mourn over the graves of lost offspring, or sigh on beholding the sickly appearance of those who survive." Of them the adults, and especially the females, suffer under hourly increasing morbific influence, and are destined at length to succumb, far within the average limits of longevity that would have been accorded them by any insurance agency in England. On the contrary, when means permit, every sacrifice is made by parents in India, under the name of "*education*," to send their children homeward, that their constitutions may become *retempered* before they are once more exposed to deleterious intertropical influences. It is a significant fact, says Nott, on whose authority these statements are made, that the oldest purely English regiment in India, the "Bombay Tufts," notwithstanding that marriages with British females are encouraged, has never been able, from the time of Charles II. to the present hour, to rear, from births in the corps, *boys* enough to supply its musical band with drummers and fifers. The same rule holds good with the Dutch in Batavia and other Indian islands. In Algeria, also, the French are beginning to find out that, unless the indigenous Arab or Kabyle will plough the fields for them, *colonization* is hopeless (BOUDIN). At Madagascar the Frenchman, and at Sierra Leone the Englishman, die off in any prolonged attempt at *colonization* (BRYSON). With the Negro races the same phenomena are observed. The Negro is evidently killed by cold. He is the native of the hottest region of the globe, where he goes naked in the scorching rays of the sun, and can lie down and sleep on the ground in a temperature of 150° Fahr.; but if removed from such latitudes to places beyond 40° north, he steadily deteriorates, and ultimately becomes exterminated. The statistics of New England, New York, and Philadelphia prove this, where the mortality is double that of the

white population (NOTT). The acclimation of the Negro has been overrated. They never, according to Dr. Nott, became proof against intermittents and their sequelæ in America.

There is, however, an acclimation, although an imperfect one, against moderately high temperature; and it is also equally true that persons who have gone through this process, and more especially their children, when grown up, are less liable to violent attacks of paludal fevers, when exposed to them, than fresh immigrants from the temperate regions. The course of disease upon fresh arrivals—new-comers—is more rapid, as a rule, and more severe.

The statistics of Sir Alexander Tulloch and Dr. Graham Balfour show very strongly that certain races cannot become acclimated in certain realms, though they may in others, far removed from their original birthplace. British soldiers and civilians, for instance, enjoy even better health at the Cape Colony than in Great Britain; while the Negro in most regions out of Africa, whether within the tropics, as in the Antilles, or out of the tropics, as at Gibraltar, is gradually being exterminated; and "before a century has passed, the Negro race will almost have disappeared from the British Colonies in the West Indies." In 1817 one of the West India Regiments, composed of Negroes, was sent to Gibraltar, and formed part of the garrison there for twenty-two months; the mortality in it was at the rate of 62 per 1,000 of the strength annually, or above four times that of the European troops in the same garrison during the same period (BALFOUR). The American statistics of the United States confirm these conclusions (CARREY, DE BOW, NOTT).

Individuals of races pre-eminent among all others for capacity for colonization and acclimation are seen in all parts of the world combating successfully climatic influences, mainly by adapting themselves to new conditions, carefully regulating their mode of life, manners, and customs, so as to suit the climate in which they live. This is the first and main point to be attended to in attempts at acclimation. Food, clothing, and habits must be regulated so as to conform to the wants of the climate of adoption. The *Caucasian* races, particularly the *Semitic*, exemplified in the *Jewish* tribe, appear to be capable of enduring all, or almost all, varieties of climate from Sweden to Aden. The *Gipsies* are another remarkable instance; but it is difficult to say whether

they should be classed as *Caucasian* or not. Probably they are not, but are *Turanians* Caucasianized in language merely (BEDDOE).

Of the *European* races the most southern, who are generally darker, seem to have a wider range of acclimation than the *Teutonic;* and of races *not Caucasian*, the *Chinese* appears to be highly favoured in this respect. The facts most favourable to the acclimation of the *Teutonic* races are the rapid multiplication of the *German Colonies* in Southern Brazil, and of the *Cape Dutch* in the hot and dry interior of South Africa. It is still doubtful whether our race will not degenerate in Australia, though at present it is remarkably healthy there, owing principally to the absence of malaria. Most of the *Dark races* suffer greatly from *phthisis* when removed to this country. Some of them seem to be inferior to the *Negroes* themselves in this respect (BEDDOE).

In the Island of Ceylon, during a series of years, the comparative ratio of mortality has been noted among five different races of which the troops are composed; and the following are the significant results, illustrating the statements now made from the Statistical Report on Ceylon, by the late Sir Alexander Tulloch and Dr. Balfour:—

	Annual deaths per 1,000 men.
Natives of Bengal and Madras,	40
Troops recruited on the Coast of Ceylon,	23
Malays,	25
Negro Troops,	50
English Troops,	69

The most minute and reliable information we possess regarding the influence of tropical climates on European races is to be found in the Statistical Reports of Colonel Sir Alexander Tulloch and Dr. G. Balfour, the works of Sir Ranald Martin, already frequently noticed, and lastly, the *Report of the Royal Commission on the Sanitary State of the Army in India*, 1863. A report by the two first-named writers, which includes the stations of Western Africa, St. Helena, the Cape of Good Hope, and the Mauritius, shows that in Western Africa, on the average of eighteen years, every soldier was thrice under medical treatment annually, and nearly half the force annually perished; and when the mortality was at its height, three-fourths of the troops perished annually. About 300 white troops were landed at different times in 1825, and in detachments: nearly every one died, or was shattered in constitu-

tion; and what is remarkable, during the whole of this dreadful mortality, a detachment of from forty to fifty black soldiers of the 2nd West India Regiment lost only one man, and had seldom any sick in the hospital. No length of residence acclimates the whites in Africa; on the contrary, it tends to their extermination. In like manner, it has been shown that the *native* troops on the Bengal establishment are particularly healthy, while the imported English are the reverse. The Report on India shows most forcibly that much of the sickness and mortality in that country is due to the unhygienic practices which prevail, and to the unsanitary conditions which are suffered to exist in the towns, cantonments, and bazaars of India—where the *causes* of disease are in "a state of absolute perfection, but which have been removed with entire success in this country." There can be no acclimation to causes of disease; and climate, *per se*, has been made to play the part of a scape-goat for the neglect of sanitary precautions ever since we set foot in India. "Every evil from which British troops have suffered has been laid at its door" (SIR CHARLES NAPIER).

These stern realities must one day or another be grappled with face to face. With the existing provisions for the sanitary state of our army, "the field of distinction in India has hitherto been a great grave-ground—a British Juggernaut." But, as Miss Nightingale justly observes, "there is not a shadow of proof that India was created to be the grave of the British race. The evidence, on the contrary, is rather in the other direction, and shows that all that the climate requires is that men shall adapt their social habits and customs to it; as, indeed, they must do to the requirements of every other climate under heaven." The Science of Medicine and Sanitary Science teach us that much may yet be done; and the voice of public opinion will insure that the recommendations of the Commissioners contained in the Report (*Report of the Royal Commission on the Sanitary State of the Army in India*) be eventually carried out. The great work of civilization in India has yet to be commenced. The climate of the East merely exaggerates the causes of disease which exist there, and which have been fostered rather than obviated by the military economy of our troops. The annals of warfare and of campaigns teem with abundant illustrations which show that the humanizing influence of the Science of Medicine is not sufficiently appreciated by the commanders of

armies. Lord Macaulay, in his *History of England*, observes of William Henry, Prince of Orange, "that it was too much the habit even of the most humane and generous soldiers to think very lightly of the bloodshed and devastation inseparable from great martial exploits; and the heart becomes steeled, not only by professional insensibility, but by that sterner insensibility which is the effect of a sense of duty." From the days of Nero, the most ferocious, to those of Lord Raglan, the most humane commander that ever lived, this historical truth has been sufficiently verified. The sufferings of the soldier are apt to be forgotten and lost sight of in the excitement which attends our brilliant martial achievements. The humanizing influence of the medical profession may yet effect a change for the better, when it enjoys a higher position in the organization of the army than it has hitherto done; when a high standard of professional education is demanded of the student of medicine who would enter the service; when the head of the medical department of the army shall have a higher official position than has yet been accorded to him; and when his representative in the field shall have a seat in those Councils of War, and a voice in that assembly, where the sanitary position of an army is always in danger of being compromised.

Persistent Pernicious Influence of Malarious Climates.—Sufficiently authenticated examples are now on record, which prove the persistent pernicious influences of malaria, both on races of mankind and on bodies of men subjected to their influences for periods of time beyond six or eight weeks, and especially during a temperature above 60°. It is known that after the Walcheren expedition our troops continued to suffer from paludal fevers for five, six, eight, and eleven months after their return to this country, although located in as salubrious quarters as could be procured for them. M. Boudin, also, in his *Lettres sur l'Algérie*, fully shows the persistent pernicious influence of paludal poison on the French and English colonists there. During the recent war against Russia the persistent pernicious influence of the residence of the troops in Bulgaria, during a period of three of the hottest months of the year, continued to make itself more or less manifest throughout the whole of the campaign in the Crimea.

The more chronic and enduring influence of marsh malaria on

a race finds abundant illustration amongst the people who inhabit Campagna, Maremma, Pontines, and other insalubrious localities in classic Italy; and in France, in Forez, La Brenne, Sologne, Berry, Dombée, and La Bresse.

Every page of Sir Ranald Martin's classic work *On Climate* goes to establish the belief in the *degrading* influence of the Indian climate, *where malaria prevails*, on European constitutions. The slow, increasing influence of a fever which no acclimation overcomes maintains the high death-rate; and Twining also long since noticed that, "*after careful inquiry, he was unable anywhere to find a sample of the third generation from unmixed European stock.*" Thus upon European colonization, where malaria is permitted to exist, in India "Nature has set her ban—a blighting interdiction"—till the progress of sanitary measures shall make this fine country the great garden of the world.

The characteristics of degeneracy, as shown by paludal races and inhabitants of such regions, may be shortly summed up as follow:—A more or less intense state of cachexia, stunted growth, engorgement of the chief viscera, especially of the spleen, languor, and inertia of all the functions—aggravation of ordinary diseases, the superaddition to them of lesions only explicable by the atony and diminished power of re-action of the nervous centres, and finally, a diminished longevity. The influence of the degenerating principle in the sphere of the intellectual and emotional faculties is no less remarkably manifested by the torpor of intelligence, the apathy, a kind of habitude, passing on under some circumstances to a state of idiocy, and under all to the most extreme indifference (MORELL, and Reviewer in *Med.-Chir. Review*, January, 1858, p. 82).

The length to which this volume has extended will not permit of further remarks on the Geography of Disease. Enough, perhaps, has been written in illustration of some of the more important directions which the study may take, and to show that the subject presents a vast field for investigation, and claims the united exertions of every one in its exploration, who is interested in the progress of the Science of Medicine, of Politics, and of Social Health. The immediate object of the study is to ascertain the laws by which disease is distributed or propagated, or the manner in which certain conditions inimical to health are found to prevail in certain localities or regions, with a view to the prevention of

disease by sanitary measures; and how many of the phenomena relative to the regional distribution of disease are elucidated by the facts of *physical geography.*

The sources of information on the subject exist in reliable tables of sickness and mortality, a knowledge of the physical conformation of the earth's surface, and the meteorological agencies to which it is exposed. A *statistical* branch has now been created in the Office of the Army Medical Department of this country, "thus at last affording an official recognition of the value of statistics in the investigation of those questions on which the efficiency of our army so much depends, and by the satisfactory solution of which so much sickness and mortality among soldiers may be prevented, and economy both in life and in money attained."

As the sun proceeds northward in the ecliptic, so the sickly season advances from the southern to the northern islands. In the Mediterranean the mortality is doubled in the hot season between July and October; and in the Northern States of North America the posts of the army are regularly abandoned as the hot or sickly season approaches. In temperate regions this order is reversed. Throughout Europe generally, the maximum mortality occurs at the end of winter, and the minimum in the middle of summer. The Registrar-General of England calculates that a fall of the mean temperature of the air from 40° to 4° or 5° below the freezing-point destroys from 300 to 500 of the population of London. The agency of the wind is manifested in the distribution of heat and moisture, and in the comparative density of the air, as well as by its direct influence as a distributor of malarial poison. The absence of wind was uniformly noted as a concomitant of cholera, which, in Britain and elsewhere, has been observed to be developed, and to be most virulent, when the calm was the greatest, and often began to abate when the wind rose.

It may not be out of place merely to indicate some definite and practically important subjects of study relative to this interesting department of medical science:—

1. A study of the climatology and the diseases of the different quarters of the globe illustrate most clearly that the morbid conditions produced by certain pathological states, while they are of a fixed character, are more intense in their severity, more continuous in their development, and more prolonged in their existence, in some places than in others.

2. A study of the climatology of towns, and such circumscribed districts, is of the greatest practical importance to the physician in all questions relative to change of air for the invalid. On this subject the work of Sir James Clark *On Climate*, with its inestimable series of meteorological tables, is the classic source of reference; while many monographs have also been written of late on particular localities.* The reader will also consult with advantage an admirable account of the effects of change of climate on diseases of the lungs, by Dr. Walshe, in the appendix to his work *On Diseases of the Lungs.*

3. Our medical officers of health are industriously mapping out the realms of disease, which often too definitely manifest themselves amongst the vilest purlieus of our cities. Systematically and energetically, many of the causes of diseases are thus more effectually exterminated. Those who have to care for the sanitary state of our armies now believe that similar means of preventing disease may be successfully applied abroad. Sanitary officers are now appointed to great military expeditions, and a board of sanitary officers is recommended, and has been established, in all the Presidencies of India. For this purpose a study of the geographical distribution of diseases, and of the causes which lead to their special distribution, obviously becomes of the greatest practical use. By it we learn that certain classes of diseases, rather than others, are mainly under the influence of terrestrial and meteorological causes—namely, those of the ZYMOTIC class, and which are stamped with special *miasmatic* characters. In proportion, therefore, as we become capable of knowing that particular diseases of this class are limited to certain portions of the earth, and can trace the meteorological laws of their geographical distribution and diffusion, we necessarily obtain clearer conceptions

* Of these the following may be referred to:—

Diseases of the New Zealanders, by Dr. Arthur Thomson. *Medico-Chirurgical Review*, commencing April, 1854.

On the Climate of Algiers, by Dr. Arthur Mitchell. *Medico-Chirurgical Review*, commencing January, 1856.

A Comparative Inquiry as to the Preventive and Curative Influence of the Climate of Pau and other Places, by Dr. Alexander Taylor.

Change of Climate, and an Account of the Climates of Spanish Towns, by Dr. Francis.

On the Climate of Spain and Australia, by Dr. Burgess. *Medico-Chirurgical Review*, October, 1854.

Climatology of the United States, by Blodget.

of their causes and modes of propagation, as well as more practical knowledge of the means of their prevention and of cure.

By such knowledge large masses of men may be more successfully cared for in foreign countries; and the topographical position and construction of habitations may be determined upon with a certain definite knowledge to guide the chooser. Military barracks and hospitals may be judiciously provided for abroad, and with all the aids of scientific knowledge, the diet, the clothing, and the military exercising of the troops may be arranged so as to suit the physical climate of the place in which armies are to campaign or garrisons to be located.

CONCLUSION.

In bringing these volumes to a close (for the *fifth* time), the Author is still painfully conscious of many sins of omission and of commission in the execution of his task. Much has been left unwritten "of the thousand ills that flesh is heir to," for which it might have been desirable to find a place in these pages; but if the work shall merely serve as a guide to the acquisition of knowledge, and the acquirement of practical skill in the Art of Medicine, the Author will have accomplished his design.

After the student has read and carefully studied all that is contained in books of this kind, he will still find that he has only made a commencement of the great study and real labour of his professional life. To extend his knowledge, and to acquire practical skill, he must possess, cultivate, and foster, above all things, the faculty of observation, combined with the exercise of a sound judgment. From such a work as this, which simply transmits to the future a sketch or record of our present knowledge of the natural history of well-defined diseases and their remedies, he may acquire a *book-knowledge* of the Science of Medicine; and, by clinical instruction, with such a book as his guide, and experienced clinical teachers to point out to him the best methods of investigation, he may also acquire some *practical experience.* It is such a method of study which will give the young medical man an extensive view of the Science of Medicine; and at the same time, with exactness of knowledge, some experience in the art of investigation, as well as in the direct and intelligent management of disease. It is exactness of knowledge which alone can give directness and intelligence of purpose in the treatment of diseases; and such knowledge will eventually place him in the best position to begin the practice of his profession.

It has been well observed by a veteran physician—Dr. Joseph Bullar, of Southampton—that "the treatment of disease is an ART requiring daily and hourly practice; and nothing but actual practice, with the responsibility on a man's own shoulders, can make a practical man." As this practice requires close attention, reflection, and judgment, as well as prompt action, the more a man's

mental powers are educated by his college training, the better a practitioner he must in time become, if he devotes himself to his duties. Those who succeed in a marked way in acquiring a knowledge of the more theoretical parts of medicine, and who show their power strikingly in obtaining a full and exact knowledge of the natural history of diseases, and of the literature of the Science of Medicine, have sometimes been set down as not likely to be practical men. But this is an unjust conclusion—a popular fallacy. It requires only time and the proof of success to show that, when such men apply their powers to practice, they become the most successful physicians. They bring to the bedside of the sick, minds well stored with professional knowledge — vigorous, quick, and well-cultivated mental powers, which cannot fail to place them amongst the most useful and trustworthy of practical men, in the application of remedies for the cure or relief of diseases—the chief end and object of all our study.

APPENDIX TO FIFTH EDITION.

On Diphtheria, (p. 568, Vol. I.)

On this subject I received a note from Dr. Wade, of Birmingham, dated January 12, 1868, expressing a hope that his "views on the relation of *albuminuria* to *diphtheria* [at page 568 of this Edition, and page 567 of Fourth Edition, Vol. I.] may be more accurately represented," and offering to point out in what respects they are incorrectly given. As the subject of *diphtheria* had been printed off previous to the receipt of Dr. Wade's letter, I could only express my regret at any incorrectness of representation, and offer to publish (in the form of an Appendix) a statement, to be furnished by himself, in his own words. Dr. Wade has therefore referred me to his notes on *Diphtheria* in the *Lancet* of 1862, and has kindly sent the following statement on the subject:—

"When my attention was first turned to diphtheria, the doctrine of Bretonneau was paramount, that diphtheria is a local disease, and for the most part always remains so, infecting the system, if at all, only by absorption of putrescent matters from the throat, and, consequently, all treatment was to be local. I was struck early by the fact that many slight cases die; and I resolved to make a full dissection as soon as opportunity should offer, without any preconception as to what I should find. In the first case I found (after about seven days' illness) a pair of white kidneys, such as one finds after scarlatinal dropsy. The spleen was occupied by a similar deposit. After this I turned my attention to the kidneys during life, and found not infrequently *albuminuria*. I have never said (because I never believed) that *albuminuria* and *uræmia* were convertible terms; but I do say that I have seen many cases of diphtheria which presented distinct symptoms of uræmia—comatose or comatoid phenomena—coincidently with an obvious diminution of the urinary secretion, and relieved (and that very suddenly) by restoration of the secretion. I looked upon the discovery of renal complication as important, for two reasons,—*Firstly*, because it showed that diphtheria does not spread solely (as Bretonneau taught) by continuity of surface. *Secondly*, as accounting for death in *certain* cases previously inexplicable.

"From considerations flowing mainly from the discovery that the 'continuity of surface' theory was erroneous, I revived the doctrine that diphtheria was an essential fever, and hence the inutility of local treatment, at that time universally carried out, under the influence, and as a corollary to, the theory of its being a local disease.

"In Dr. Sanderson's essay, he seems to suggest that I had overlooked the fact of there being hyperoxidation in diphtheria. So far from ignoring it, it formed the basis of my views.

"Dr. Sanderson relates a case in which there was, with *albuminuria*, abnormal amount of renal excretion, and says that this case proves that my views are

incorrect, and that in no case does insufficient elimination exist. I suggest that, *in the first place* (admitting all the facts of this case to be as he has stated), to draw the inference that, in no case can there be insufficient elimination is to commit the logical error of reasoning from a particular to a universal. *In the second place*, admitting that the renal elimination going on during the height of the pyrexia was vastly greater than that of a period of health or convalescence when the ingesta were greater, proves nothing at all as to the adequacy of the elimination during the former period. The real question is this—'*Was the elimination adequate to remove from the system the abnormally excessive quantity of effete material produced by the pyrexia?*' To this Dr. Sanderson's comparative experiments give no answer. There are no scientific data in existence which can answer it. I admit that it is eminently desirable that these should be procured. But this is to be done, not by comparing the total daily excreta during convalescence with those of a part of the pyrexial period, but by comparing the excreta of the '*pre*-renal' pyrexial period with those of the '*post*-renal.' In the meantime, the answer I offer to the question is based upon clinical experience. I find a certain amount of urine, of a certain specific gravity, *without* certain general symptoms one day, and the next day I find urine of less specific gravity, probably less in total quantity, *with* certain general symptoms. These symptoms resembling some of those found in cases of pure kidney disease (and commonly reputed to depend upon that disease), I infer that, in these cases also, the symptoms depend upon insufficient elimination. I do not say that we find these symptoms in all cases of diphtheria. I do not say even that we find them in all cases of diphtheria with albuminuria.

"As regards treatment, I recommend the adoption of the Cullenian doctrine here, as in other specific pyrexiæ—namely, 'to avoid the tendency to death.' One tendency is to death by insufficient elimination of effete material. I try to avoid this *principally* by copious supplies of aqueous liquids. I do not object to meet other tendencies by appropriate remedies, whether of a stimulant, tonic, or any other suitable character.

"I also strongly condemn the indiscriminate employment of topical applications, as being most painful and distressing to the patient, and commonly inoperative. On the other hand, some of the most striking recoveries I have seen have been unquestionably due to the timely use of local means suitable to the exigencies of the particular case."

On the Subject of Worms, (p. 871, Vol. I.)—

Dr. Heslop, of Birmingham, wrote me (also on the 12th of January), to say that the account of the *cerebro-spinal* symptoms of worms is remarkably defective, and kindly sent me part of an essay of his on the subject, originally printed in the *Dublin Quarterly Journal*, No. 55, from which the following conclusions are reproduced:—

"1. That in the great majority of cases of tape-worm, and, though with lesser frequency, in cases of other intestinal worms, more or less serious and peculiar nervous disturbances are apt to arise.

"2. That the most frequent of these are headache, giddiness, various troubles of the special senses, especially singing in the ears, flashes and dark spots before the eyes, imperfect amaurosis, and trembling of the limbs.

"3. That various anæsthetic and, on the contrary, neuralgic phenomena are

very frequent, usually connected with general lassitude and sense of muscular feebleness.

"4. That, though less frequent than those previously cited, convulsive seizures, partaking of the nature of epilepsy or acute eclampsia, or sudden attacks of insensibility, mixed with syncope and, in the female sex, severe forms of hysteria, are also often directly traceable to worms.

"5. That the last symptoms (No. 4) are more common in childhood and the earlier periods of life than afterwards, and are more frequently caused by the round and thread worms than by tape-worm.

"6. That chorea does not appear to be often excited by the irritation of worms.

"7. That a feeble state of the general health generally accompanies the presence of worms; often in cases of tænia, proceeding to marked anæmia, so as even to lead to the suspicion of the possible existence of Bright's disease.

"8. That the irritative phenomena of the digestive tube, even when associated with various symptoms referred to the functions of that tract, do not warrant the diagnosis of the presence of tænia; and that their absence does not absolutely indicate the absence of the parasite.

"9. That the frequent appearance of the nervous symptoms above related, without a well-marked relation to any special lesion of the nervous system, especially if alternating with periods of perfect or nearly perfect freedom, should engender the suspicion that worms are present. If to these symptoms are added various ill-defined disturbances of the functions of assimilation, including occasionally colicky pains, without marked vomiting, pain after food, or decided emaciation, it is in the highest degree probable that worms are the source of the symptoms.

"10. That it is probable that many of the symptomatic phenomena of vermination are connected, not with their direct irritation of the mucous membrane with which they are in relation, but with a general disorder of the system, partly resulting from the parasites, and partly the cause of their maintenance and development in the intestinal tract."

LATHYRUS SATIVUS, (p. 860, Vol. I.)

Further accounts of this vetch as a cause of paralysis may be found in *Indian Annals of Medical Science,* vol. vii., 1861, by the late Dr. Kinloch Kirk, p. 144; also by Dr. Irving, pp. 127 and 501. An incidental reference is also made in Thomson's *Travels in the Western Himalaya and Tibet,* p. 391, footnote.

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